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The Surgical Treatment of Acquired Heart Disease Utilizing Extracorporeal Circulation*

• Surgical technics and cardiopulmonary bypass, previously employed with success in the treatment of congenital heart lesions are now applied to the correction of acquired heart disease. Case reports are given of some of the excellent work done by the Department of Surgery of Tulane University.

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WITH the exception of rheumatic mitral stenosis, treatment of acquired heart disease has been notably ineffective largely because of inability to correct valvular dysfunction under direct vision. The successful results obtained in the treatment of congenital heart lesions utilizing cardiopulmonary bypass have been followed logically by the extension of these techniques to the treatment of acquired heart disease. The use of an extracorporeal circuit to bypass the heart and lungs has made it possible for the surgeon to determine at operation the type and extent of valvular abnormality and to carry out a precise correction of the deformity. This report is concerned with our experience in the treatment of acquired heart disease utilizing a pump-oxygenator for cardiopulmonary bypass.

Special Diagnostic Procedures

In order to evaluate a patient with acquired valvular heart disease from a surgical standpoint, it is essential that the diagnosis be confirmed and the extent of physiologic derangement produced by the valvular abnormality determined. Thus, in addition to routine roentgenographic and electrocardiographic studies, catheterization of the right and the left heart chambers is necessary. The technic for right heart catheterization is standardized and is routinely employed in most institutions when involvement of the mitral valve is suspected. Catheterization of the left heart chambers, however, has been developed more recently and there are several technics available. In our laboratory two procedures have been employed,

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namely, the transbronchial, left atrial puncture method described by Allison and Morrow [1] and the transthoracic left ventricular puncture method described by Sir Russell Brock. In the former, a needle is inserted under bronchoscopic control through the antero-inferior wall of the left main bronchus into the left atrium. A catheter is then passed through the needle into the left atrium and pressures are recorded successively from the left atrium, left ventricle, and ascending aorta. These data are confirmed by pullback tracings recorded as the catheter is removed. By this technic the diagnosis and quantitation of mitral stenosis, mitral insufficiency, aortic stenosis, and aortic insufficiency are determined. This procedure has proved safe and relatively free from complication.

For measurement of the pressure gradient across the aortic valve in patients with aortic stenosis, the transthoracic left ventricular puncture is extremely useful. A number 20 gauge needle 6 inches in length is introduced through the anterolateral aspect of the left chest into the left ventricle. With a needle in the left brachial artery, pressures can be measured simultaneously from the left ventricle and the systemic artery and the gradient across the valve determined. This technic is simple and can be carried out rapidly but its associated complications are more frequent and serious than are those occurring with the use of the transbronchial technic.

Acquired fistulas between the aortic sinuses and the heart chambers may be detected by cardiac catheterization if the right heart chambers are involved or by angiocardiography if the left heart chambers are affected. Cinefluorography or serial roentgenography with injection of contrast material through a catheter passed into the ascending aorta via the brachial artery will clearly establish the diagnosis in either instance.

The following case reports illustrate the use of these special diagnostic methods and the corrective surgical technics applied in patients with acquired heart disease.

Aortic Valvular Disease

Case No. 1.—J. M., a 41-year-old man, was admitted to Touro Infirmary on March 28, 1958. He had had occasional attacks of syncope on exertion extending over a period of six years. These attacks were infrequent until six months preceding admission when they began to appear after mild exertion and were accompanied by severe precordial pain relieved only by complete rest and nitrites. There was no history of rheumatic fever.

On physical examination blood pressure was 110/70. There was a harsh systolic murmur heard over the entire precordium with maximal intensity at the aortic valve area and transmitted along the great vessels to the neck. The point of maximum impulse was forceful and displaced downward into the left anterior axillary line.

Chest roentgenogram revealed left ventricular enlargement with calcification of the aortic valve. Electrocardiogram was indicative of left ventricular enlargement and this was associated with frequent premature ventricular contractions.

On March 25, 1958, catheterization of the left ventricle was performed, and revealed a gradient across the aortic valve of 100 millimeters of mercury.

Operative exposure of the aortic valve was performed on April 1, with the aid of extracorporeal circulation and elective cardiac asystole induced with potassium citrate solution injected into the coronary circulation. The orifice of the aortic valve was only 8 millimeters in diameter (Fig. 1). Despite heavy calcification, it was possible to identify the commissures. These were incised with heavy scissors and the opening was then dilated to a diameter of 1.5 centimeters. With careful resuscitation the heart resumed effective contraction and the operation was terminated.

Postoperatively the patient developed excessive fibrinolysis and bleeding occurred into the anterior mediastinum. This was corrected by administration of fibrinogen, fresh whole blood, and evacuation of the anterior mediastinal hematoma. He was discharged on April 18.

Case No. 2.—A 36-year-old man was admitted to Charity Hospital, on July 25, 1957, with a diagnosis of aortic insufficiency. Two years previously he experienced constricting substernal pain while performing heavy labor. The symptoms were relieved by rest and recurred with resumption of activity. When advised of his condition he restricted his activity and remained asymptomatic until six months before admission when symptoms recurred with increasing severity. The administration of nitroglycerine and digitalis produced relief of symptoms. One month before admission following prolonged physical

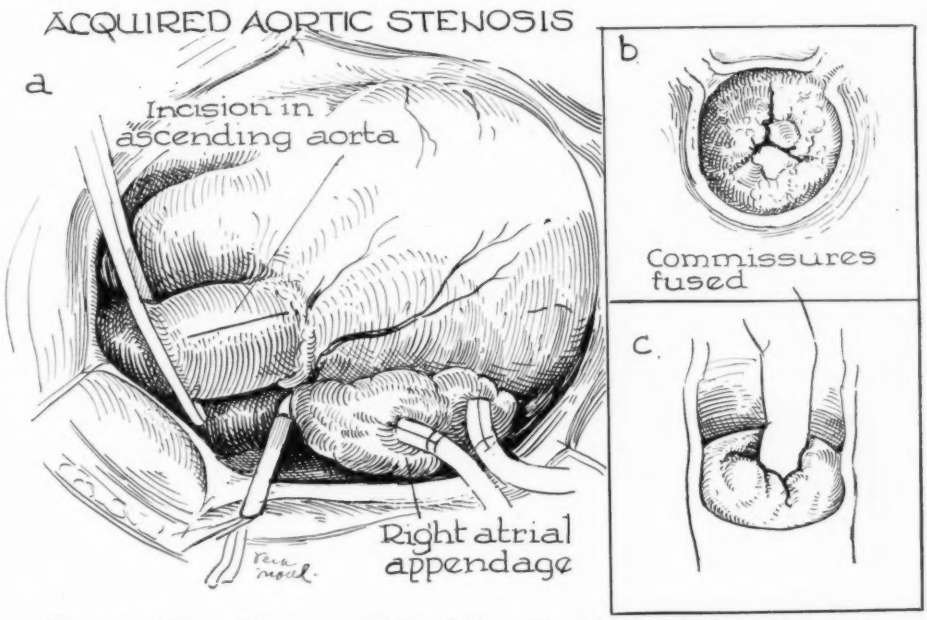

Figure 1.—Diagramatic representation of the aortic valve in calcific aortic stenosis.

activity a third attack of intense chest pain developed which required bed rest. In 1929, the patient was ill for about one month with prolonged fever. He was rejected for military service in 1943 for unknown reasons. There was no history of joint pains or venereal lesions.

Physical examination revealed vigorous nodding of the head with each cardiac contraction, visible heaving of the precordium, and visible pulsations of the subclavian triangle. The heart was enlarged downward to the anterior axillary line at the sixth intercostal space. There was a grade 3 basal systolic murmur widely transmitted particularly into the neck; and a systolic thrill was palpable over the neck vessels. A grade 2 diastolic murmur was loudest in the third intercostal space along the right sternal border and was transmitted unchanged to the apex. Auscultory blood pressure in the right arm was 176/30/0 mm. mercury and was the same in the left arm. Pressure in the lower extremity was 340/80/0.

Roentgenograms of the chest revealed cardiac enlargement and widening of the ascending aorta (Fig. 2). In the electrocardiogram there were wide QRS complexes with inversion of T_1, T_2, T_3, a high RV 5 and the presence of Q waves in leads 2 and 3. These findings were interpreted as evidence of myocardial disease, left ventricular hypertrophy and myocardial ischemia.

On August 8, 1957, operation was performed utilizing extracorporeal circulation. Through a bilateral anterior thoracotomy in the right fourth and left third intercostal spaces with transection of the sternum the intrapericardial portion of the aorta was exposed. The aortic annulus was exposed by mobilization of the right atrial appendage and the atrial wall in contact with the posterior surface of the aorta. The aorta was mo-

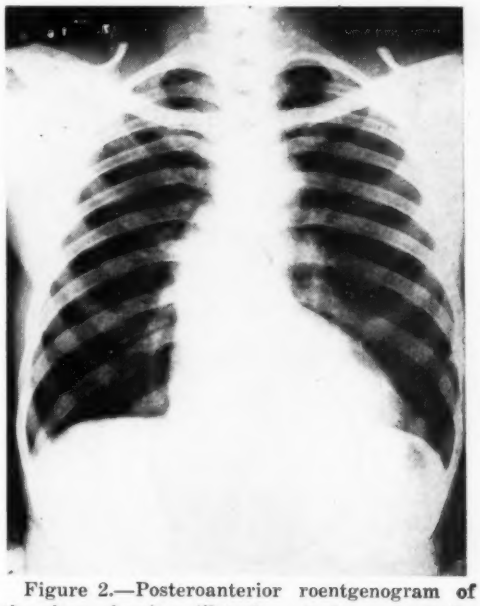

Figure 2.—Posteroanterior roentgenogram of the chest showing dilatation of the root of the aorta and the cardiac configuration of aortic valvular incompetence.

bilized from the annulus to the origin of the innominate artery and a tape passed about it. After extracorporeal circulation was instituted the ascending aorta was cross-clamped and the heart was stopped by injection of potassium citrate.

An incision was made in the aorta extending into the posterior sinus of Valsalva. Exploration of the valve revealed normal aortic cusps but a severely dilated annulus and widening of the commissures. It was noted that insufficiency was occurring at the commissures as well as in the central portions of the lumen where the cusps failed to coapt because of dilatation of the annulus. Using mattress sutures reinforced with bolsters of Ivalon the noncoronary sinus of Valsalva was obliterated thus reducing the aortic diameter about one-third. Thus the right and left leaflets were approximated on the folded posterior cusp which projected into the lumen to provide additional competence (Fig. 3). The aortic incision was closed and coronary circulation re-established. Cardiac action began promptly and a regular sinus rhythm was resumed without difficulty (Fig. 3). The total period of perfusion was thirty-four minutes and cardiac arrest twenty-seven minutes.

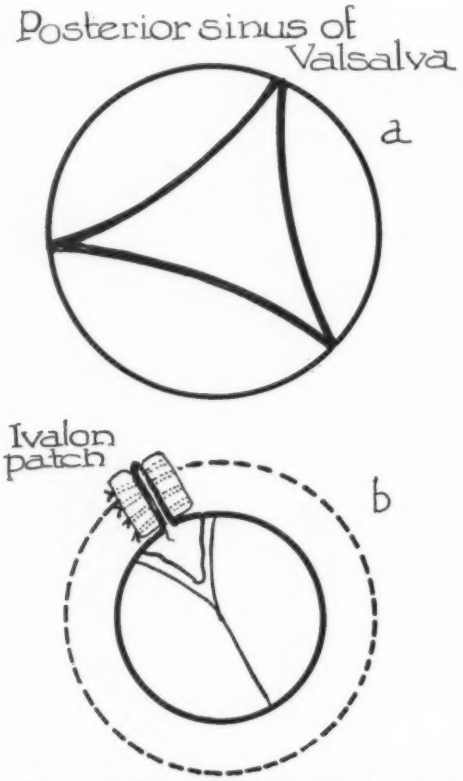

Figure 3.—Diagram of the reconstruction of the aortic annulus and aortic valves.

The patient was discharged three weeks after operation with auscultory blood pressure in the upper extremity of 136/65. Two months after operation he was re-admitted to the hospital where it was noted that the hemodynamic changes resulting from aortic insufficiency were significantly diminished. Ausculatory blood pressure was 150/70 in the upper extremities. Twenty-one months after operation the patient works as a farm hand and has had no recurrence of chest pain.

These two cases are examples of the use of extracorporeal circulation in the management of lesions of the aortic valve. There is disagreement regarding the relative merit of closed and open approaches to the aortic valve, particularly in the treatment of aortic stenosis. However, we believe that direct vision correction of the stenotic valve is desirable. Experience with six cases suggests that even in the presence of markedly calcified cusps, one or more commissures will be partially or completely fibrous thus permitting effective commissurotomy.

Since it is possible to perform commissurotomy within five to eight minutes, we have found potassium arrest unnecessary and now simply cross clamp the ascending aorta for the period necessary for manipulation of the valve. The resulting myocardial ischemia is well-tolerated. In fact, the heart continues to beat although the rate may be slowed.

Any patient who has already begun to show enlargement of the left ventricle and who has either dizziness, syncope, or angina on exertion as a result of aortic stenosis, should be considered for surgical correction. At this stage of disease, surgical intervention can be undertaken with little operative risk. Once left ventricular failure has occurred or generalized congestive failure is present the risk of any surgical procedure is increased significantly.

The surgical treatment of aortic insufficiency began in 1953, with the introduction, by Hufnagel, of a plastic ball valve. This valve has been used extensively since then with symptomatic relief in many instances. However, the operative mortality has been about 15 to 20 per cent and

correction of valvular incompetency is only partially accomplished because of the necessity of placing the valve in the descending aorta. A number of investigators have attempted to improve the function of the incompetent aortic valve by constriction of the aortic annulus, but these procedures have proved unsatisfactory. We have felt that in many cases of aortic insufficiency the cusps are relatively normal, insufficiency resulting from dilatation of the aorta at the level of coaptation of the valves. The procedure described above has the advantage of utilizing the natural valves and of restoring competency at the root of the aorta where it is most effective. It appears that this case represents the first instance in which this technic has been utilized.

Acquired Aorto-Cardiac Shunts

Case No. 3. — A 26-year-old man had been stabbed in the chest during a fight on April 1, 1956. He was operated upon at Milwaukee County Hospital and a wound in the right ventricular outflow tract was closed. At this time, because of a systolic and diastolic thrill, a diagnosis was made of penetration of the ventricular septum with a left-to-right shunt; however, the patient was in satisfactory condition following closure of the wound in the right ventricle so nothing further was done. Three weeks later cardiac catheterization revealed an abnormal communication at the level of the right ventricular outflow tract with a left-to-right shunt of 1.7 liters per minute.

In March 1958, the patient appeared at the Tulane Medical Clinic complaining of exertional dyspnea and easy fatigability and requested that the abnormal communication within his heart be closed.

Physical examination revealed a bilateral, anterior thoracotomy scar in the fourth interspace. The heart was normal in size but a systolic thrill was palpable at the base. There was a continuous "machinery" murmur heard best over the left, third intercostal space near the border of the sternum.

Roentgenographic examination of the chest revealed increase in pulsation and prominence of the pulmonary vessels. An ECG was normal.

On March 27, a fistula between the aorta and the right ventricular outflow tract was closed under direct vision with the aid of extracorporeal circulation. The patient tolerated the operation well, and he was discharged from the hospital within two weeks.

Case No. 4.—A 27-year-old housewife was admitted to Charity Hospital on January 7, 1958, complaining of exertional dyspnea, frequent attacks of tachycardia and palpitation of two years' duration. She had known of a heart murmur for three years but had been asymptomatic.

In July 1957, while in the third trimester of her first pregnancy, she suddenly developed congestive heart failure. Symptoms responded to the administration of digitalis and there was an uneventful delivery in September 1957. However, symptoms persisted after delivery and became more pronounced until her admission to the hospital in January 1958.

On examination a systolic thrill was palpable over the upper anterior chest and a soft systolic murmur was heard at the base of the heart. In addition, there was a harsh diastolic murmur along the left sternal border extending as far as the apex of the heart. The heart did not appear to be enlarged.

A chest roentgenogram was normal although fluoroscopic examination demonstrated slight enlargement of the left ventricle. The electrocardiogram was normal.

Cardiac catheterization, performed on January 9, 1958, demonstrated a left-to-right shunt at the atrial level. These findings were indicative of an aortic-atrial fistula secondary to rupture of a sinus of Valsalva.

On March 14, operation was performed. The thorax was opened through a bilateral anterior thoracotomy in the fourth intercostal spaces, transecting the sternum. The right atrium was greatly enlarged. There were numerous fibrotic excrescences on the surface of the right atrium and at the site of these areas it was possible to palpate a powerful jet of blood striking the atrial wall. This jet could be traced back to the portion of the right atrium in contact with the root of the aorta. By pressure through the atrial wall at this site the jet could be completely stopped and the thrill in the atrium eliminated. Using cardiopulmonary bypass and potassium arrest of the heart, the right atrium was opened widely and the orifice of the fistula was visualized in the upper medial portion of the right atrium. The mouth of the fistula was calcified so that it was impossible to close the opening by means of sutures. Therefore, a plug of compressed Ivalon was inserted into the fistulous tract and sutured to the margins of the defect with several silk mattress sutures. (Fig. 4).

The postoperative course was complicated by tachycardia which was finally identified as being secondary to hypopotassemia and digitalis intoxication. This was corrected by the administration of potassium and withdrawal of digitalis

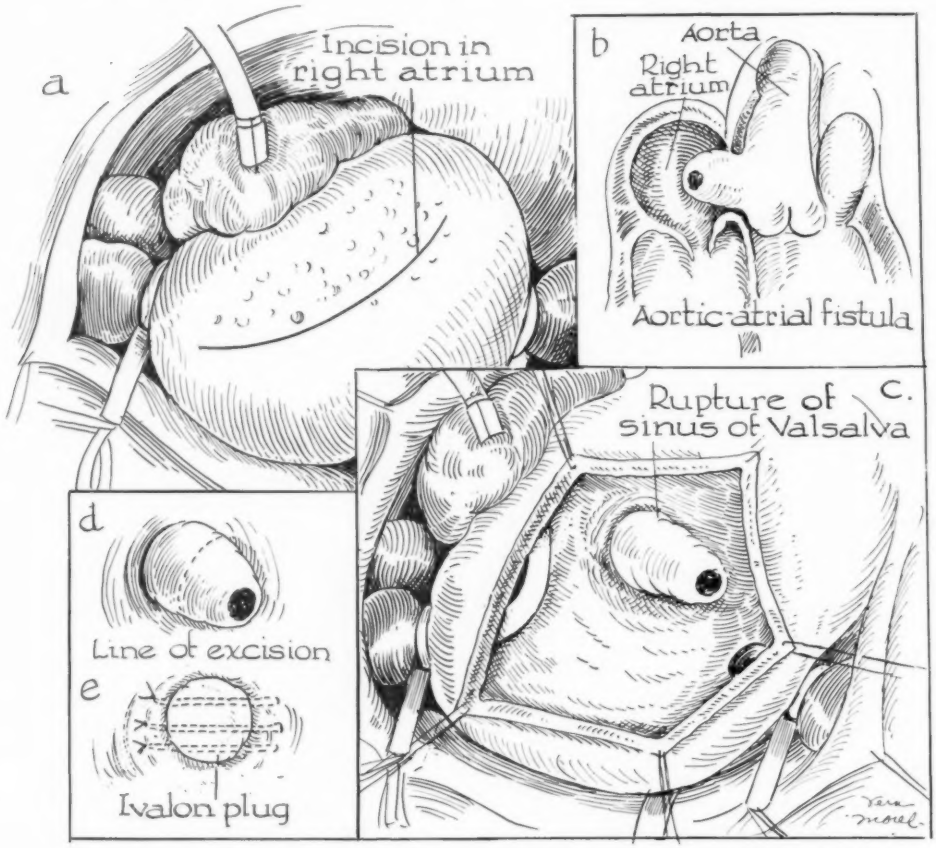

Figure 4.—Diagramatic representation of operative repair in aorto-atrial fistula.

and she was discharged on April 9. At the time of her last clinic visit she was asymptomatic and carrying on her household activities.

Previously a case of traumatic defect of the ventricular septum successfully closed with the aid of extracorporeal circulation was reported.[2] The case described here is the second traumatic intracardiac shunt which we have treated. In this patient a traumatic fistula between the aorta and the right ventricle developed following a stab wound of the heart. A similar case was recently reported by King and Shumacker.[3]

These cases of traumatic intracardiac shunt are of considerable interest and are perhaps not as uncommon as was formerly supposed. Many of these patients undoubtedly die as a result of their initial injury. In those who survive there is little tendency to spontaneous healing of the intracardiac shunt.

Aorto-atrial fistula secondary to a ruptured aneurysm of the sinus of Valsalva is being reported with increasing frequency. While the essential defect is a congenital deficiency of the elastic layer of the aortic wall at the level of the annulus fibrosis, clinical symptoms do not arise usually until the aneurysm ruptures either into one of the cardiac chambers or into the pericardial sac. In the older literature, rupture of the aneurysm is described as a dramatic event, accompanied by severe substernal pain not unlike that of myocardial infarction with the subsequent development of severe congestive failure resulting in death within a short time. It is now apparent that rupture of the aneurysm may not be a sudden catastro-

phe at all. The patient reported here was known to have a murmur resembling that of aortic insufficiency for several years, and the fistula must have been present during this entire period. The diagnosis of an intracardiac shunt at the atrial level was made by means of cardiac catheterization and with the clinical manifestations suggestive of aortic insufficiency, a correct preoperative diagnosis of an aortic-atrial fistula was possible.

In this patient the fistula was obliterated by a modification of a method described by Morrow [4] in which the opening in the root of the aorta is occluded by a snugly fitting tampon of Ivalon that is sutured to the margins of the defect. This appears to be a satisfactory method of dealing with this lesion. The rapid subsidence is indicative of complete elimination of the fistula.

Mitral Valve Disease

Uncomplicated mitral stenosis is best treated by closed, digital commissurotomy and it is unlikely that open methods will supplant this technic any time soon. On the other hand, open technics using cardiopulmonary bypass may be indicated for certain mitral valve abnormalities. These are as follows: (1) stenosis after an adequate commissurotomy; (2) mitral stenosis with recurrent embolization and auricular fibrillation; (3) mitral stenosis in conjunction with an atrial septal defect (Lutenbacher syndrome); (4) significant mitral insufficiency with or without mitral stenosis.

Not all cases of recurrent mitral stenosis need be subjected to open operation. As emphasized by Harken there has been progressive improvement in the technics of mitral commissurotomy and it is possible that some of the patients treated several years ago by closed methods would be benefited by another closed procedure utilizing the improved technics.

In mitral insufficiency on the other hand, it is our experience that the closed technics of repair are unsatisfactory. Carefully selected patients with mitral insufficiency can best be helped by an intracardiac valvuloplasty performed with the aid of extracorporeal circulation.

Summary

The surgical treatment of acquired heart disease has been expanded and rendered more effective by the use of open heart technics. Precise diagnosis utilizing catheterization of the right and left sides of the heart and special roentgenographic methods is essential. Aortic stenosis and insufficiency, fistulas between the aorta and cardiac chambers, and lesions of the mitral valve may now be corrected surgically.

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Discussion

Dr. Paul T. DeCamp (New Orleans): It is a privilege to open the discussion following Dr. Creech's excellent paper on the current status of cardiac surgery. I have elected to discuss briefly the vascular problems associated with stenosis of the major blood channels to two important organs, the kidney and the brain.

Sufficient narrowing of the artery to one or both kidneys results in severe, progressive hypertension. It occurs in early life associated with congenital strictures, and more commonly late in life associated with arteriosclerotic plaques. The diagnosis is suggested by typical arteriographic findings and certain findings of differential renal function studies. It is proved at operation by finding a significant fall in blood pressure across the area of stenosis. The disease is curable either by removal of the distal renal tissue, or by reconstructing the vascular channel. In 8 cases, we have performed nephrectomy in 3, and vascular reconstruction in 5. In every instance, the hypertension has been corrected or markedly improved, and symptoms relieved. One elderly patient died from cerebral vascular insufficiency which developed as the hypertension disappeared.

Stenosis of one or more carotid arteries in the neck characteristically produces the syndrome of "little strokes", often recurring frequently and with varying degrees of interval remission. Again

arteriography demonstrates the lesion. Bilateral carotid lesions were present in 4 of our 12 operative cases. In cases in which the vessel is narrowed, but open distally, vascular reconstruction is possible and clinically rewarding. The brain is protected during periods of operative occlusion of the vessel by hypothermia or temporary vascular bypass. In 12 cases we have performed endarterectomies in 7, in 2 instances, vascular transplantation, and in 1 case each a replacement graft, unbridling, and sympathectomy.

In 8 cases, partially occluded vessels were restored to normal function with clinical improvement in 6. An acute traumatic occlusion of the common carotid artery was corrected but death occurred from prior brain damage. In 2 cases of complete sclerotic occlusion of the internal carotid artery, vascular reconstruction was impossible, but in 1 of these improvement has followed cervical sympathectomy. In 1 instance, an embolus occurred postoperatively aggravating the disease.

From our experience we are convinced that stenotic lesions in the main vessels to the kidneys or brain may cause severe symptoms. They can be accurately evaluated by arteriographic technics, and are frequently amenable to corrective surgical therapy.

Historical Notes

We extract from **"De Bow's Commercial Review"** the following interesting reminiscence of the **"Olden Time in New Orleans and Yellow Fever:"** It was written by Maunsel White, Esq., an old and highly respectable citizen of New Orleans. **(Ed.)**

* * * * We landed our fleet of flat boats a little above Gravier, and within ten steps of Tchoupitoulas street, at 10 o'clock in the morning on the 1st of August, 1801, after a passage of 60 days from Louisville in Kentucky. The writer, then a mere lad, thought New Orleans to be a very queer looking place, and was astonished at the jargon of strange tongues. The city at that time was in the hands of the Spaniards and Don Manuel Salcedo, Governor. It was a fortified town. There was a moat or ditch cut around it, and an embankment with palisades and several small redoubts placed at convenient angles. These works were in a state of decay at that time, and were of no defence to the city. They ran from the river where Canal street now is, back to Rampart street, thence down to Esplanade street, and from Esplanade to the river, below where the mint now stands. There were three gates, through which people passed from 6 o'clock in the morning until 9 o'clock at night, when they were shut. At each was placed a sentry box with a corporal's guard. In those days of Spanish gate rule a corporal was an important man, and marched before the Governor, when he took his evening's walk on the levee, to clear the way should any one dare approach him, without a low bow, or without leaving a wide berth. The free and easy Kentuckians were particularly obnoxious, as they could not comprehend why a mere man in the shape of a Governor should be any better than themselves, and looked with astonishment at the deference paid by the Spaniards to such a decrepit old fellow as Governor Salcedo then was.

New Orleans M. & S. J. 5:370 (November) 1848

Traumatic and Surgical Shock*

• As the author quotes: "Shock is easier to recognize than to describe, and easier to describe than to understand." It is a subject with many facets, and a thorough exposition such as the author gives still leads him to conclude that there is much to learn with regard to the basic physiology of shock and the supportive care of patients in shock.

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SHOCK is probably the most common of the serious problems among surgical and seriously traumatized patients. The term is not a precise one and is generally applied to the clinical syndrome characterized by progressive circulatory imbalance as the body responds to injury or noxious stimuli. Further definition is noted in the stages impending, reversible, and irreversible, and additional classification is obtained in describing the initiating or precipitating factor, such as (a) primary circulatory pathology due to medical, neurological or even psychiatric disturbances; (b) biological factors, including bacteria, chemical, metabolic toxins; and (c) blood or plasma depletion due to trauma, wounds, hemorrhage, surgery, peritonitis, obstruction, perforated viscus, exposures to heat, cold, and irradiation and electrolyte disturbance.

As Dr. Truman Blocker has stated "shock is easier to recognize than to describe and easier to describe than to understand." The most characteristic findings are rapid pulse of low volume, low blood pressure, pallor and sweating, even though the skin may be dry and warm, subnormal temperature, shallow depressed respiration, apathy, reduced sensibility and motor weakness, apprehension, restlessness, excessive thirst, scant urine.

Mechanism

The older theory of the mechanism of shock indicated that reduction in blood volume produces decreased delivery of oxygen to the tissues, generalized increase in capillary permeability from anoxic damage, and loss of plasma from the minute vessels to reduce further the circulating blood volume. The newer theory, particularly of Wiggers, subject to much controversy because of the conflicting experimental evidence, would indicate that many factors are at work. Wigger's interpretation is that a reduction in circulating volume results in decreased venous return, decline in cardiac output and default of the peripheral vascular circulation due to: (1) loss of vasomotion in the viscera with stagnation and pooling; (2) portal stasis from increased hepatic and splenic resistance; (3) marked reduction in renal flow due to persistence of renal constriction which occurs along with constriction of skin vessels as a compensatory mechanism in the initial and reversible stages of shock.

Reduction in blood volume, whether actual or relative, leads to a decrease in the venous return, which produces a fall in the pressure of the right atrium and central veins. The ventricles adjust with a small systolic discharge in a more rapid heart beat. The body then compensates, attempting to increase the circulating blood volume and to improve the venous return by vasoconstriction of surface and renal vessels, by increasing the rate and depth of respiration and perhaps by contracting the spleen to provide autotransfusion. If these compensatory maneuvers are not sufficient, the arterial pressure falls in spite of the vasoconstriction, and

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so-called stagnant anoxia occurs in selected tissues, particularly the liver and kidney.

This leads us to consider the vasomotor excitor material VEM, which is apparently formed in the anoxic kidney. This factor in turn seems to be integrated with the adrenal cortex and with the sympathetic nervous system activity. Also a vasodepressor material VDM is formed and also inactivated by the anoxic liver. This factor has been defined as the iron protein, ferritin. These factors then are thought to be a part of the mechanism which, after initial phase of lowered pressure and rapid pulse of impending shock, leads very rapidly to a state of vasoconstriction as a compensatory mechanism. There is subsequently a vasodilating influence in which much blood is pooled, sequestrated or stagnated in sections of the body, particularly in the splanchnic areas. As a matter of fact, the circulating blood volume may be depleted primarily by by-passing a considerable section or segment of the blood volume, particularly by a vaso-excitory influence on the precapillary sphincter.

As decreased effective blood volume occurs, anoxia results and the VDM factors liberated either by excessive or incomplete inactivation in the liver are set forth. The chain of events may then continue on into the irreversible or critical oligemic state with ultimate circulatory collapse. In this final clinical manifestation of shock, the ultimate causes of irreversibility are yet to be determined. They may be concerned with liver, pituitary, adrenal hormones, toxic degradation products from extensive tissue damages, accumulation of metabolites as the result of anoxic changes or bacterial invasion from the gastrointestinal tract.

With this brief introduction to the shock syndrome let us pass on to the more specific consideration of surgical and traumatic shock. Hypovolemic shock includes in its classification, — wound shock, surgical shock, operative shock, traumatic shock, burn shock, crush injury shock, hemorrhagic shock, and in addition, shock associated with water loss or water deprivation (chronic illness resulting in decreased blood volume, and infections such as peritonitis). However, hemorrhage is the most frequently encountered cause of this type of shock.

Decreased Blood Volume

Management of hypovolemic shock in less severe traumatic and surgical problems may be anticipated and prevented. In this respect proper evaluation of the patient preoperatively is quite important. This includes those points in the history which might contribute to a decreased blood volume. Cancer patients, debilitated and malnourished patients from gastrointestinal disease, and patients with chronic weight loss present the clinical picture of decreased blood volume in spite of perhaps normal red blood counts as well as hematocrit readings. It is in these patients that blood volume measurements, such as those which determine the total red cell mass or the plasma volume itself, as determined by the Evans blue dye test or by chromium tagged red cells or by the I^{131} radioactive studies, may be more beneficial than at any other time. Serial determinations are especially valuable and in combination with the clinical features present a fairly reliable index as to how much blood should be given preoperatively. It is much safer and wiser to give blood prior to surgery than to expect to replace it and compensate for contracted blood volume on the operating table. Therefore blood repletion should be carried out twenty-four to forty-eight hours prior to surgery.

These same principles may be fairly well applied to the less severely traumatized patient. It may be possible by history to anticipate that the injured individual has, prior to surgery, had a contracted blood volume. In addition, it may be expected that the injury itself has created only a moderate blood loss. Under such circumstances early replacement of blood may prevent a fissure progressing to the need of a great deal more blood than otherwise might have been needed.

Blood Replacement

If the depletion of the circulating blood volume is sufficient to proceed on to true surgical or traumatic shock the corner stone of therapy is blood replacement. Transfusion of whole, properly cross-matched blood, is the best form of therapy. Patients who exhibit minimal evidence of shock, that is, a drop in the pressure and an increase in the pulse rate, have lost their minimum of 20 percent of their circulating blood volume and, as the shock deepens, an estimation of 40 to 75 percent of the blood volume is lost. Early replacement of 1000 to 1500 cc. of whole blood may effectively bring the patient out of the classification of beginning shock.

No specific formula can be given as to the amount of blood needed. The systolic blood pressure should be kept to 100 millimeters or above and the pulse rate to 120 or less and the urinary output up to 30 cc. or more per hour.

Experiences in World War II and the Korean War, demonstrated the fact that 3 to 5 pints of blood generally will be required if the systolic pressure is below 80 millimeters; and if 40 percent or more blood loss has occurred, massive amounts of blood may be safely given and needed to reverse the process of seemingly irreversible shock. Many of the Korean casualties received from 30 to 40 pints of blood within twenty-four hours and in some instances within ten hours.

In the surgical patient it may be possible to estimate the amount of blood lost by measuring aspirated material, by the procedure of counting sponges or by the plan of weighing sponges. It is generally stated that each 4 by 4 sponge may represent an average of 10 cc. and a lap. sponge may be estimated to contain 100 cc. These methods may be as much as 50 percent in error, and therefore, need to be replaced with more blood than is estimated. In the traumatized patient, tremendous losses of whole blood may occur in compound fractures and as much as 2 or 3 liters may be lost around simple or closed fractures of the femur. Multiple fractures, especially involving the spine or pelvis may cause tremendous loss of blood, as of course can readily occur in the chest and abdomen. Rapid replacement of whole blood with possible consideration of low titer O-positive blood in the beginning and then matching after the first one or two units may prevent the series of events that lead to a more progressive shock.

First Aid Measures

Restoration of the traumatized patient should begin with appropriate first aid measures to stop hemorrhage, splint fractures, provide an adequate airway; close sucking wounds; conserve body warmth; and intravenous sedation for pain but not for restlessness due to anoxia. This treatment should be concomitant with the restoration of the circulating blood volume by whole blood. And almost concomitantly it must be realized that surgery itself may be a part of the resuscitative measure in controlling the bleeding.

Surgical Intervention

Surgical intervention should be planned in those cases of traumatic shock in whom 1000 cc. of whole blood does not resuscitate the patient and should be planned at about the time of the completion of an additional 500 cc. of blood. If one can be sure that all bleeding has been controlled, it is well to give 500 cc. more blood after the systolic blood pressure has exceeded 100 millimeters. In those cases where surgery is anticipated and shock is not present but the extent of the wounds or injuries indicate rather significant blood loss, at least 500 cc. should be given before starting anesthesia. Blood may be given at 2 or 3 portals simultaneously and may be given intra-arterially but at present this particular method does not seem indicated except in exceptional instances. It has been observed in these instances that intra-arterial blood seemed to be more effective than intravenous blood perhaps through perfusion of the brain and heart more directly and possibly by by-passing the blockage effect

of the lungs. After blood has been restored the vein should be kept open by the use of 5 percent dextrose and water before and during surgery and continued where fluid and electrolyte balance requires. This can best be followed by maintaining a urinary output of around 40 cc. per hour.

Hazards of Transfusion

In discussing replacement of blood volume with whole blood, one must emphasize the need for blood but at the same time remember some of the hazards of transfusion. Of course mismatching and typing errors are foremost among the causes for serious reactions from blood transfusion. This is rarely a laboratory error but nevertheless mislabeling and the failure to identify properly the blood to be given and the patient to receive, have resulted in catastrophes.

Hepatitis also results from whole blood transfusion and it has been stated that about 1 donor in every 150 is believed to be a carrier of the hepatitis virus and that he cannot be detected by screening measures or laboratory tests. It is true, however, that only 1 in 4 recipients is susceptible to the disease should they receive the blood, therefore, the attack rate among patients receiving blood, is about 1 in 600 or an incidence of 0.2 to 0.5 percent.

Citrate intoxication is another problem associated with massive transfusions. This reaction may occasionally cause concern but if one gram of calcium gluconate is administered as a routine after each liter of blood, when rapidly administered, this danger is minimized. Further, the problem of developing bleeding tendencies is of some importance but also can be fairly well controlled with the calcium gluconate administration.

It has been wisely stated that if blood is needed in the surgical patient, more than one unit is generally indicated. A single transfusion makes no significant improvement in the anemia and is too little for a real therapeutic agent in severe hypovolemic shock. It is to be remembered that blood is the surgeon's great ally, but it must be wisely used.

Plasma Volume Expanders

Plasma expanders are to be used in emergency while awaiting the availability of whole blood. A commonly used form is 6 percent Dextran, a product developed by Sweden, and first made in England, and prepared in such a way that the average molecular weight of its fractions is in the general range of serum albumin. This material is nonpyrogenic and nonantigenic and does not appear to be dangerous to the kidneys.

Polyvinylpyrrolidone (PVP) is a synthetic product prepared by the Germans during World War II. It is made from acetylene and formaldehyde, and is quite effective in its osmotic expansion of the plasma volume. Its chief drawback appears to be that only about 65 percent of it is excreted from the body, the rest apparently remaining indefinitely and more particularly stored in the liver. It is an inexpensive product in comparison with Dextran and so far as is known is not hepatoxic even after prolonged and careful study.

A third plasma expander, ossein gelatin, is an excellent product with none of the apparent drawbacks so far as the body physiology is concerned and one that remains in the circulation a desirable length of time but has the disadvantage of a high gel point, in that it does not remain liquid except when stored in a warm place. It is perhaps the safest of the plasma volume expanders, and would seem to be very suitable for use where its high gel point does not make a practical difference.

In using pooled plasma and in using concentrated albumin solutions, one encounters several advantages and a good many disadvantages. For example, in pooled human serum there is at least a theoretical and perhaps a practical possibility of virus hepatitis in 20 percent. This incidence of hepatitis has not been affected by exposure to ultra-violet light but has been improved greatly by storing

the plasma at room temperature for a six months' period. The addition of beta-tropriolactone seems to further reduce the possibility of serum hepatitis. Human serum albumin is quite effective but of course is quite expensive since it requires about 3 or 4 average blood donations to yield the 25 grams of albumin ordinarily dispensed in 100 cc. of fluid. The plasma volume expanders serve a useful purpose in resuscitation, and can be used effectively up to 1000 or 1500 cc. volumes.

A special use of the plasma expanders is concerned with traumatic shock in which there is loss of plasma in excess of the amount of loss of whole blood. Examples of this problem may be seen in trauma causing gross soilage of the peritoneum or pleura with intestinal contents in abdominal and thoraco-abdominal wounds, in cases of gas gangrene of the edematous type, and, of course, in burns. A switch to a plasma expander can be made after administering 1000 to 1500 cc. whole blood if it is thought proper that they should be alternated so as to decrease the bleeding tendency created by dilution of some of the blood clotting mechanism.

Fluid in Burn Therapy

In burn therapy the total amount of fluid predicted to be needed in the first twenty-four hour period is obtained by multiplying the weight in kilograms by the percent of body burned times three. Of this total fluid, half should be administered as colloid bearing solutions, including blood. Twenty-five percent should be administered as isotonic saline, and the remaining 25 percent as dextrose and water. Of course if the burned patient is able to tolerate liquids by mouth the total volume of oral intake may be subtracted from the intravenous need so far as the noncolloid bearing solutions are concerned. Such an oral fluid would do well to contain a teaspoon of sodium chloride and one half teaspoon of sodium bicarbonate or sodium citrate.

Cardiorespiratory System

Primary attention has been focused on the replacement of whole blood in the management of surgical and traumatic shock and mention has been made of the necessity of treatment of the wound at the same time as the shock treatment is under way. It is necessary to be sure that a clear airway is obtained, that a sucking wound of the chest be occluded, that a tension pneumothorax be relieved and that secretions be removed from the respiratory system even if tracheostomy be necessary. These measures supersede even the control of hemorrhage. And of almost equal importance to the relief of these problems is the control of the hemothorax, pneumothorax, hemopericardium or cardiac tamponade. Additional supportive measures should be included. Chest pain may be a big factor in continuing the shock and should be controlled by appropriate pain relief, particularly by nerve block. Partial relief of cardiorespiratory dysfunction may be obtained by Levin tube, oxygen therapy, as well as optimum position.

Differential Diagnosis

In traumatic shock it is wise to be alert to the differential diagnosis in those cases in which shock or peripheral circulatory failure must always be differentiated from acute cardiac failure, cardiac tamponade, hemorrhage or emphysema in the mediastinum obstructing the superior vena cava, pulmonary embolism, poisoning from barbiturates, alcohol or morphine, coma from head injuries and adrenal insufficiency. Also chlorpromazine, that is, thorazine and phenergan may be incriminated in postoperative or post-traumatic hypotension.

Vasoconstrictors

For the most part there is very little controversy about the standard measures for the control of surgical or traumatic shock. However, the question of vasoconstrictors, antibiotics and cortisone is more controversial.

The vasoconstrictors are not generally needed where blood replacement is needed. They are primarily used in that type of shock due to vasodilator influences. However, all cases that proceed to the type

of shock described as irreversible, but not proved to be irreversible until death actually occurs, should receive vasoconstrictors. Of these norephinephrine (Levophed), methed metaraminol (Aramine), neo-synephrine and mephentermine (Wyamine) have received the widest use. They are of especial benefit in treating shock due to myocardial infarction, bacteremia, hypersensitivity reactions, and those forms of shock thought to be neurogenic in origin. Levophed and Aramine, particularly, have a direct beneficial effect on the myocardium, increasing the contractility and increasing the cardiac output. Both of these drugs display the vasoconstrictive effects of epinephrine, but neither produce cardiac acceleration, stimulation of the central nervous system, or depression of the appetite.

Levophed has the disadvantage of being extremely transient in its effect, and therefore, is to be given only intravenously and needs careful minute by minute observation. Aramine may be injected intravenously, intramuscularly, or subcutaneously, and has a more prolonged period of action. Aramine also is supposed to produce less renal constriction than Levophed. Levophed administered can be varied in its concentration according to the needs in maintaining the blood pressure adequately. Tremendous dosages have been used without obvious side effects of toxicity. Aramine can be given with a single intravenous injection of 3 to 10 milligrams and can be given at one half to two hour intervals or can be given subcutaneously and be expected to last as long as ninety minutes. Such a vasoconstrictor should ideally mimic the selective action of the body's own reflexes. None of the drugs available do just exactly this. However, Levophed does produce a marked vasoconstriction and is effective in raising the blood pressure in shock when other agents have failed and does not decrease renal function as expected from studies in the normal individual. It does have the disadvantage in normal individuals of decreasing cerebral blood flow; however, it is not known to do so in shock, but is thought from an elevation of the blood pressure to increase the cerebral circulation.

Antibiotics

Another factor of controversial importance in the management of shock is the administration of antibiotics. Much experimental work, particularly that of Fine, has indicated an endotoxin probably arising from the intestinal tract as being of great importance in producing irreversible shock. It was also observed in the Korean Campaign that the use of one half to one million units of penicillin and streptomycin intravenously was thought to be beneficial in resuscitation of advanced shock patients. Dr. Fine states in reference to the role of infection in traumatic shock, that it is his theory that when death occurs in traumatic shock in spite of proper therapy, the probable cause is a bacterial toxin which produces irretrievable collapse of the circulation. Furthermore if there is no systemic or local infection to account for the toxin, an endotoxin derived from the intestinal bacteria is the most likely offending agent. He supports this statement with experimental work and seems to feel that irreversibility is prevented by administering the antibiotic prior to onset of shock.

Sepsis is perfectly capable of producing shock and in its clinical manifestations there is a triad consisting of sepsis, shock, and hyperthermia. The terminal picture of uncontrolled infection is often that of total biological break-down, that is, circulatory and respiratory decompensations accompanied by renal shut-down, liver failure, jaundice, fever, shock and coma finally occurring. Treatment of shock and infection involves the consideration of accurate blood volume replacement, vasopressor therapy, total body cooling and oxygen therapy.

Corticosteroids

Another ancillary agent in the treatment of resistant shock is the use of the corticosteroids. There is no clear evidence that adrenal insufficiency usually is involved in the causation or progression of

shock; however, the stress reaction may precipitate a sequence of events which leads to the initial diagnosis of Addison's disease, and of course those patients who have been on corticosteroids and those patients who are chronically debilitated with stress syndrome, are all potential candidates for beneficial effects from cortisone therapy. Therefore, in all patients who have not responded to other forms of therapy, intravenous hydrocortisone in very heavy amounts is worth a trial.

It is thought that these drugs definitely enhance the effectiveness of vasopressor agents and suppress inflammation and particularly suppress the systemic reaction to inflammation. Such therapy would have a particular place in surgical patients in relation to extensive tissue injury. Its use may provide a brief period of grace in which normal circulatory adjustment may be re-established and specific treatment instituted.

The following program is suggested in reference to management with corticosteroids:

1. Increase the amount of corticosteroid preoperatively if the patient has been receiving such therapy, never cease or decrease.
2. Increase the salt intake during and after surgery. (If salt loss is occurring, 6 to 9 grams of sodium chloride may be necessary for the daily requirement).
3. Estimate blood loss accurately and administer these amounts accurately. (If the patient is sweating provide extra salt replacement).
4. Treat arterial hypotension by intravenous use of hydrocortisone.
5. Watch for signs of fluid retention by palpating the patient's bony prominences.
6. If increased stress, such as postoperative complication occurs, increase the amount of salt and of cortisone if necessary.
7. As soon as the salt balance can be obtained and maintained orally, this is the preferable form.
8. Maintain good nutrition even if necessary to give testosterone.
9. These patients should be watched closely for metabolic and electrolyte disturbances.
10. Asthenia, lassitude, weakness, arterial hypotension, indicate impending adrenocortical crisis. A low CO_2 combining power, excessive urinary salt loss, low serum chloride and sodium, should also alert one to the need for treatment.
11. The urinary output should be measured and charted daily and if more than 2,000 cc. it should be suspected of containing an excessive amount of salt loss.
12. If the patient has some cardiac disease associated, watch this carefully and correct with the proper therapeutic aid.

Other Measures

Another ancillary method of treatment is the use of hypotensive drugs, sometimes described as the hibernation treatment of shock. It has been shown experimentally that chlorpromazine reduces the mortality of animals following hemorrhage of injection of endotoxin. It would appear that in some instances prior treatment with the drug may be of some value in increasing the resistance to shock.

Briefly, anticoagulants may be mentioned as of some benefit in shock therapy, increasing the chance of survival in those patients in whom thrombosis in small vessels may be prevented. Also the use of hypothermia which is primarily used in cardiac surgery should be mentioned as should the artificial kidney. Dialysis may tide the patient through the oliguric state following shock.

Summary

In summary, a brief review of some of the facts and theories concerning the pathogenesis of shock and more particularly of surgical and traumatic shock, has been presented.

Emphasis has been placed on the restoration of a normal circulating blood volume and the correction of disturbances of the cardiorespiratory function.

Once the problems of external bleeding, impaired respiratory function, cardiac tamponade, et cetera, have been corrected,

further management of shock consists of a fairly well established group of principles plus certain newer theoretical and more controversial ancillary measures.

We still have much to learn with regard to the basic physiology of shock and the supportive care of patients in shock. There is no substitute however, for practical experience and we have learned from army experience in the recent war that many patients could be saved from irreversible shock by a refusal to accept the diagnosis of irreversibility and by extending our courage and ingenuity in treatment.

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Paroxysmal Ventricular Tachycardia*

• The author discusses the etiology of this condition, the diagnosis and the clinical and electrocardiographic signs of differentiation, as well as the prognosis, and complications, and outlines the important aspects of adequate treatment.

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PAROXYSMAL ventricular tachycardia is an arrhythmia of idio-ventricular origin. The ectopic focus may be in either ventricle. There is usually a slower auricular rhythm, although any auricular rhythm may be present. In contrast to supraventricular tachycardia most cases of paroxysmal ventricular tachycardia occur in significantly diseased hearts.

Etiology

In about 75 to 85 per cent of the attacks of paroxysmal ventricular tachycardia there is underlying heart disease; the most common type being atherosclerotic coronary disease with or without hypertensive heart disease. Many cases of paroxysmal ventricular tachycardia develop following myocardial infarction. Harris [13] et al concluded from their experiments on dogs that the paroxysmal ventricular tachycardia associated with infarction is due to release of significant amounts of potassium from the ischemic and infarcted muscle. Wasserman and Yules [3] report a case of paroxysmal ventricular tachycardia associated with a cardiac aneurysm following an infarction. A number of cases [6] have been precipitated by the two step exercise tolerance test in patients with a very mild angina. The next most common type of heart disease present is rheumatic heart disease. Ventricular tachycardia seems to have no predilection to being associated with any particular valvular damage in rheumatic heart disease.

A number of reports have pointed out that ventricular tachycardia may occur in normal hearts. Most of these cases have been associated with emotional upset or nervous tension. Ring and Blankfein [4] reported many recurring attacks of ventricular tachycardia in the same patient over a nine year period all associated with emotional strain and fatigue. Psychodynamic factors were so important in one case that the patient was finally relieved of his paroxysmal ventricular tachycardia by psychoanalysis after heavy doses of quinidine and procaine amide had failed.

Several drugs play a prominent part in producing paroxysmal ventricular tachycardia and the upsetting thing about this is that some of these same drugs are recommended in the treatment of ventricular tachycardia. Baker [5] et al reported a case of P.V.T. due to quinidine therapy of auricular fibrillation. Digitalis as a cause is well known. In Herrmann's [8] series, 10 per cent of the patients had signs of digitalis intoxication and 64 per cent of the total number were taking digitalis. Paradoxically, Baker [5] et al thought the interruption of their cases was due to Digoxin therapy.

Diagnosis

The symptoms of paroxysmal ventricular tachycardia are similar to those of other tachycardias; except that the condition is more frequently associated with severe heart disease, and as a result, the symptoms are more pronounced. They usually consist of a disturbing, rapid heart action associated with a feeling of weakness, which may progress to collapse and prostration. There is usually marked anxiety and dyspnea and, on occasion, a tightness in the chest. There may be a relative

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coronary insufficiency with angina, or coronary thrombosis may develop.

The physical signs in ventricular tachycardia are of considerable value in separating this arrhythmia from tachycardias of other origin. The ventricular rate usually ranges from 120 to 200 beats per minute. In minute to minute counting of the pulse, there is frequently a slight irregularity with rates differing from 5 to 7 beats per minute. Schrire and Vogelpoel [2] pointed out that this slight variation is hard to recognize and also may occur in supraventricular tachycardia, particularly, auricular flutter. Ventricular tachycardia does not respond to vagal stimulation by carotid sinus pressure, Valsalva maneuver, or other methods. These procedures usually change the rate in supravenrticular tachycardia.

Much [2, 7, 8, 21] has been written about the changing intensity of the first heart sound in paroxysmal ventricular tachycardia. This is a very valuable sign, but frequently is difficult to recognize. The variation in the first heart sound is due to the disassociation between the auricles and ventricles. The major component of the first heart sound is due to the closure of the mitral and tricuspid valves. The position of the auriculo-ventricular valves at the beginning of systole is paramount in determing the intensity of the first heart sound and is related to the PR interval. During ventricular tachycardia, the PR interval is continually changing and thus varying the intensity of the first heart sound. Of course, if auricular fibrillation is present or if there is retrograde conduction to the auricles, there is no variation in the first sound.

Harvey [7] has emphasized the multiple staccato type sounds that one hears in paroxysmal ventricular tachycardia. These sounds are due to the wide splitting of the first and second heart sounds and gallop sounds. The splitting of the heart sounds is due to the asynchrony of the ventricular contractions in P.V.T. Splitting of the first sound is best heard at the mitral area and lower left sternal edge. Splitting of the second sound is best heard at the pulmonary area and the third left intercostal space. In tachycardia, the first sound is grossly accentuated but the second sound is diminished. As a result, splitting of the first sound in P.V.T. is usually much more easily recognized. Of course, in a supraventricular tachycardia with bundle branch block, there is splitting of the first and second heart sounds; but it is usually not as marked as in ventricular tachycardia and there is no variation in the intensity of the first heart sound.

The occasional giant waves in jugular pulsation in paroxysmal ventricular tachycardia are of considerable diagnostic importance. These large waves are produced by the contraction of the right auricle on a closed tricuspid valve. With the exception of nodal tachycardia with retrograde blocking of nodal impulse giving rise to A-V disassociation, irregular large jugular "A" waves occur only in ventricular tachycardia.[2] Of course, when auricular fibrillation is present or there is retrograde conduction to the auricle there will be no irregular giant jugular "A" waves.

In some cases, the electrocardiographic diagnosis of ventricular tachycardia is clear cut and simple. However, in many cases the diagnosis is very difficult and even may be impossible. This difficulty is particularly manifested in separating supraventricular tachycardia with bundle branch block from ventricular tachycardia.

One of the established EKG criteria of ventricular tachycardia is the slight variation in rate. A perfectly regular rhythm, especially if the rate is over 200, suggests that the tracing may not be a ventricular tachycardia. The QRS complexes in P.V.T. are aberrant and prolongated; also, they vary slightly in contour due to the occasional supra-imposed P waves. This results from the separate, usually slower, rate of the auricles. Of course, the identification of P waves occurring independently and usually at the slower rate is most helpful. The P waves are frequently difficult or impossible to identify in

the ordinary 12 lead EKG. However, they can often be seen if V 1 and V 2 are made in the second right intercostal space. At times, esophageal leads may be the only way of identifying the P waves. In rare cases, there is retrograde conduction with P waves occurring at the same rate as the QRS, usually late in the QRS or following it. The diagnosis is supported if isolated ventricular premature complexes either antedating or following the arrhythmia can be found. Of course, these complexes must have the same form as complexes during the paroxysm; also, they are followed by compensatory pauses. If the end of the paroxysm can be recorded, it will be followed by a compensatory pause.

The presence of "capture" beats with normal QRS complexes is usually accepted as diagnostic of ventricular tachycardia. The "capture" beats occur when the S.A. node impulse happens to arrive at the ventricle just at the right time in the paroxysm to stimulate the ventricular musculature to contract. Since the beat is conducted over the normal pathways, the QRS is of normal appearance. If these "capture" beats have aberrant conduction and resemble the other complexes in the paroxysm, this is strong evidence against a ventricular tachycardia. Finding such "capture" beats may be the only way of separating A-V disassociation with nodal rhythm and bundle branch block from ventricular tachycardia.

Fisch and Pinsky [21] pointed out that finding a right bundle branch block pattern should make one suspicious that the diagnosis is not paroxysmal ventricular tachycardia. This is the bundle branch block that frequently occurs in supraventricular tachycardia when the rate goes above a certain critical level. If the onset of the tachycardia can be recorded, gradual aberrations of the QRS complexes suggest that it is a supraventricular tachycardia and not a ventricular tachycardia. Of course, vagal stimulation should always be tried during the EKG recording of the tachycardia. A positive vagal response is evidence against a ventricular tachycardia.

Prognosis

Clinicians have long been aware of the fact that the clinical courses of patients with ventricular tachycardia may vary substantially. In some cases, the patient may not even be aware of the attack of paroxysmal ventricular tachycardia while other patients may go rapidly downhill with a profound fall in blood pressure and a fatal termination. Corday [23] et al have demonstrated in dogs that there is a marked drop in the systemic and coronary blood pressure and in coronary blood flow in P.V.T. The ventricular rate did not seem to be the only factor controlling the coronary flow. At rates below 160, the site of the ectopic ventricular focus is the dominant factor. They showed that a focus at the base of the ventricle caused a much more marked reduction in aortic pressure and coronary flow than did a similar focus located at the apex. This suggests that the site of the ectopic ventricular focus has considerable bearing on prognosis and clinical course.

The prognosis in P.V.T. is influenced by many factors, such as rate, duration of attack, precipitating factors, nature of underlying heart disease and response to therapy. In prolonged attacks, myocardial failure frequently develops. Good response to therapy in these cases improves prognosis markedly. Herrmann [8] et al have pointed out the poor prognosis in patients with myocardial infarction who develop ventricular tachycardia, even when there is a good response to therapy. The mortality rate in this group in his series was 77 per cent. In contrast, the prognosis in cases with no heart disease is excellent. The over-all mortality in all P.V.T. is high. In Herrmann's [8] series of 28 cases of persistent P.V.T. there were 17 deaths within two months of onset. In all cases, there is danger of ventricular fibrillation developing. This is especially true in multifocal ventricular tachycardia, which indicates extreme irritability of the myocardium.

Complications

One of the complications of ventricular tachycardia is the frequent occurrence of postparoxysmal electrocardiographic changes. These usually consist of ST segments and T wave changes and are due to subendocardial ischemia developing during the prolonged tachycardia. These changes normally clear up in a day or two after termination of the arrhythmia; however, they may last several weeks. With prolonged tachycardia and the resulting circulatory stasis, there is a tendency for the formation of mural thrombi with ensuing pulmonary and systemic emboli. This same situation holds true in the coronary circulation, and thrombosis with myocardial infarction may occur. In long-lasting attacks, there is a high incidence of congestive failure. Myhre [16] reported two cases of Stokes-Adams syndrome occurring during attacks of ventricular tachycardia and pointed out that 51 cases of Stokes-Adams syndrome associated with various forms of ventricular tachycardia had been reported up through 1952.

Treatment

Recently [23] it has been pointed out that ventricular tachycardia may be terminated by the use of vasopressor drugs. Corday [30] et al have shown that these drugs are effective when the heart is completely denervated. This suggests that the effect is due to the elevation of the blood pressure or action of the drug directly on some anti-arrhythmic center in the heart to terminate the paroxysms. If the arrhythmia is caused by some ischemic area in the myocardium, elevation of the blood pressure may increase the coronary flow, thereby, relieving the ischemia and ending the tachycardia. Norepinephrine, intravenously, is probably the most effective vasopressor drug and is especially indicated if there is any hypotension or shock-like condition present.

The anti-arrhythmic effect of hydroxyzine (Atarax®, and Vistaril®) has been pointed out by Burrell [28] et al. These drugs can be given orally, intramuscularly, and intravenously, and seem to be relatively free of serious side effects. The above authors reported 3 cases treated with intravenous Atarax in doses varying from 50 to 100 milligrams with relief of tachycardia in all the cases in a matter of minutes. These drugs appear to act through some specific anti-arrhythmic effect on the myocardium and through their sedative or tranquilizing effects. As a prophylactic against ventricular tachycardia, these drugs given orally seem worth a try. In patients on anti-coagulant therapy concurrently with hydroxyzine (Atarax), the dosage requirement of the anticoagulant drugs was markedly decreased.

Morphine, subcutaneously or intravenously, has been effective in a number of cases. Scherf [27] reported he had used digitalis or Strophanthin in a number of cases of ventricular tachycardia and found it very effective. He does not agree with the often made statement that digitalis is contraindicated in cases of ventricular tachycardia.

Quinidine has been used both for treatment and the prevention of ventricular tachycardia. Given orally, it is probably the drug of choice except in the most severe cases. The only absolute contraindication to quinidine therapy is a history of serious reaction, such as thrombocytopenia with fever occurring during previous quinidine administration. Most authorities state that quinidine should not be used when complete heart block is known to be present but this is a relative and not an absolute contraindication. Serious toxic reactions with quinidine are rare.[26] Gastrointestinal upsets with nausea, vomiting, and diarrhea are common, and unless severe, do not indicate discontinuance of the therapy. Potentially serious is the depressing action of quinidine on the heart. Prolongation of the QRS beyond 25 to 50 per cent requires discontinuance of the drug. When quinidine administration is associated with an increase in ventricular rate or transient ventricular fibrillation, the drug should

be stopped. Oral or intramuscular quinidine occasionally causes a moderate fall in blood pressure, and this may be very marked in intravenous therapy.

Dosage schedule will vary with the requirements of the particular case. Usually, it is recommended that a test dose of quinidine be administered before starting a regular schedule of quinidine therapy. However, in a serious condition such as ventricular tachycardia, I do not believe this delay is indicated. In the average case, quinidine can be started on 0.2 grams to 0.4 grams every two hours. If normal sinus rhythm has not been restored after three or four equal two hour doses, the size of the dose should be increased by 0.10 to 0.20 grams. This schedule may be repeated until the arrhythmia is corrected or toxicity appears. It is seldom wise or necessary to give over 1.0 gram individual doses. Frequent electrocardiograms are necessary to detect the early signs of toxicity.

When a more rapid action is indicated, the drug may be given every hour and increased in the same manner as when given every two hours. Of course, on this schedule, there is a much greater risk of toxic side effects. In urgent cases, if the favorable response is not seen in one or two hours after the first dose, the next dose may be increased 0.1 gram or 0.2 grams and so on. On this schedule, there is a high incidence of toxicity and electrocardiograms should be taken frequently.

Recurrences of tachycardia are usually related to low serum quinidine concentrations. Doses of 0.2 grams to 0.4 grams, three times a day, frequently are inadequate and doses of 0.4, 0.6, or even 0.8 grams every six hours are required.

When the acute arrhythmia is associated with shock, unconsciousness, vomiting, or other factors, parenteral quinidine may be indicated. Solutions of quinidine gluconate or quinidine hydrochloride are satisfactory for parenteral administration. The usual intramuscular doses are 0.4 to 0.8 grams which may be repeated three, or rarely, four times at hourly intervals. Only under the most urgent circumstances is intravenous quinidine indicated. A solution of 0.8 grams of quinidine gluconate in 50 cc. of 5 per cent glucose can be given at a rate of 1 cc. and at the most 2 cc. per minute. However, Sigler [20] reported successful termination of a severe case of ventricular tachycardia by giving a 5 cc. vial of 0.6 grams of quinidine full strength, intravenously. The only sign of toxicity he mentioned was a transient apnea with profound cyanosis. An electrocardiogram should be taken constantly during the intravenous quinidine therapy so as to detect widening of the QRS or any other signs of toxic depression of the myocardium. The blood pressure must be watched closely as there is often a rather marked drop in blood pressure during the intravenous therapy.

Procaine amide (Pronestyl) is probably the most effective parenteral drug in the treatment of ventricular tachycardia. Its mode of action is not thoroughly understood but it seems to depress ventricular excitability. In large doses, toxic effects are rather common. Anorexia, nausea, and vomiting are frequent. Hypotension of significance occurs only rarely after oral doses. Some of these toxic effects, such as fever, various rashes, and agranulocytosis may be on an allergic [11] basis. In some cases, an eosinophilia has been reported. Antihistamine therapy has been used along with procaine amide in an attempt to control the allergic responses.

When the condition of the patient is satisfactory enough to allow a lapse of hours between initiation of therapy and the interruption of the arrhythmia, the drug should be given orally. The usual dose is 1.25 grams followed in one hour by 0.75 grams. If this dose is not adequate, doses of 0.5 to 1.0 grams every two hours may restore normal rhythm. After intramuscular procaine amide, an effective blood level is reached in fifteen to sixty minutes. Hypotension is much rarer than with the intravenous route. The [22] initial dose should be about 1.0 grams. If indicated, subsequent doses of 1.0 grams may be repeated every one

to three hours. When immediate antiarrhythmic effects are necessary, intravenous procaine amide is indicated. One gram of the drug should be diluted in 100 cc. of 5 per cent glucose and given at not over 100 milligrams per minute.[20] The total doses at one time should not exceed 1.0 to 2.5 grams. However, Embree [18] and Levine reported a case which required 4.0 grams given intravenously over a thirty-six minute period. Lovelace [11] and Walker consider the speed of injections of major importance. They successfully treated a resistant case by giving the drug at the rate of 400 milligrams a minute. Speed of injection increases both the hypotensive side effects and the risk of intraventricular block.

Due to the frequent rather marked hypotensive effects of intravenous procaine amide, the blood pressure should be taken every half minute during the injection. A vasopressor drug should be at hand for immediate use if necessary. Continuous or intermittent electrocardiographic recording during the injection is necessary to recognize any widening of the QRS. If the QRS complexes widen over 50 per cent, the injection should be stopped so as to reduce the danger of ventricular fibrillation.

Of course, if ventricular tachycardia is due to digitalis intoxication, the treatment of choice is discontinuance of digitalis, plus potassium therapy. Twenty milli-equivalents of potassium in 500 cc. of 5 per cent glucose should be given by slow intravenous drip.

Finally, Marmor and Kert [10] reported a case of ventricular tachycardia that was resistant to the usual drugs and responded only to psychoanalysis.

Summary

In this paper, I have attempted to discuss the etiology, diagnosis, prognosis, and complications of ventricular tachycardia. In addition, I have reviewed the important aspects of treatment.

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Discussion

Dr. Louis Levy II (New Orleans): I congratulate Dr. Deming on his presentation of this subject.

In regard to etiology, it is rare that this is not associated with organic heart disease. Most cases I have seen have been associated with coronary heart disease.

In regard to diagnosis, periodic accentuation of the first heart sound is difficult to determine with a rapid ventricular rate. The differentiation by ECG of this condition from supraventricular tachycardia, at times, is difficult; this includes relatively regular auricular fibrillation with aberration and also slow auricular flutter with aberration. A clue is sometimes obtained by close inspection of the electrocardiogram for an independent auricular rhythm.

In regard to therapy, I personally have had more difficulty with the use of intravenous Pronestyl than I have had with intravenous quinidine. This has been mainly due to occurence of shock reactions. I do not believe there is very often an indication for oral preparations in this disturbance when it occurs in association with myocardial infarction. In this situation I use a 1% quinidine preparation, that is, 7½ grains in 50 ccs., and I usually utilize 50 cc. in approximately five minutes. The main use of such preparations as Vistaril, Atarax, oral quinidine and Pronestyl is probably in ventricular tachycardia not associated with coronary disease, where the speed of effect is not important.

In regard to intramuscular administration, most of these patients are in shock and will not pick up the drug intramuscularly and that is why I utilize the IV route.

These are very minor additions to Dr. Deming's paper, and I wish to congratulate him on his excellent presentation.

Health of the City

In our editorial remarks for the July number of this Journal, we stated that rain fell almost daily throughout the month of June; and now we may make the same statement in regard to the months of July and August.

	Inches
June 1847	7.233
July 1847	17.885
August 1847	5.450
	30.568
June 1848	30.818
July 1848	9.242
August 1848	6.843
	46.903

If heavy and constant showers for three months can generate yellow fever, we may well look forward with painful apprehensions. But it must be admitted that the most careful observations and sapient predictions are constantly baffled in reference to the appearance of our epidemics; for we maintain that as yet we know nothing of the causes of yellow fever. It prevails alike amid daily showers or eternal sunshine.

New Orleans M. & S. J. 5:270 (September) 1848

The Present Status of Poliomyelitis In Louisiana*

• The need for laboratory studies in the diagnosis of poliomyelitis, the development of the immunization program in Louisiana, and an analysis of statistics obtained by the Louisiana State Board of Health, are very thoroughly discussed by the authors.

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DISEASES due to viruses are growing in importance in the field of preventive medicine. For a long time, the study of these diseases has been hampered by lack of methods which might be applicable for routine laboratory testing. New methods, techniques, apparatus, and approach to these diseases have been developed, and many of these are now being used in the laboratories of the Louisiana State Board of Health.

Although the epidemiology of poliomyelitis is becoming fairly well known, the dynamics of occurrence has changed during the past half century. Progress in laboratory diagnosis and prevention, however, are fairly recent developments. It is therefore pertinent that an evaluation of the status of poliomyelitis during the past decade be made. First of all, to find out whether any aspect of its known epidemiology has changed enough to need attention: second, to explore the possibility of finding new epidemiological knowledge; and third, to appraise the effectiveness of the immunization program.

The Work of the Laboratory

In the process of doing field epidemiology in poliomyelitis where precise diagnosis is the pivotal point around which the validity of the whole statistical system revolves, we are faced with the problem of laboratory confirmation. We depend here on the virus isolation and complement fixation techniques. Even fresh specimens submitted to the laboratory do not all give confirmatory results. Usually, only about 75 per cent of the specimens from paralytic cases are confirmed by laboratory methods under favorable circumstances, and from nonparalytic cases, much less. In Tables 19, 20, and 21, it will be noted that this high rate of confirmation was not attained. This was due to the fact that many of the specimens came from outlying areas and were received in the laboratory in a not too fresh state. Yet it was found that in those specimens which were taken from cases in the New Orleans Charity Hospital and immediately submitted to the laboratory, 75 per cent resulted in confirmation of the paralytic cases. Another problem is that Coxsackie and Echo viruses may produce signs and symptoms simulating poliomyelitis, and, in relation particularly to nonparalytic cases, may lead to misdiagnosis. Under present conditions, therefore, the use of poliomyelitis statistical data derived only from clinical diagnosis and collected in the routine manner poses many problems of validity. This should be remembered in evaluating the results obtained in the present study, since only part of the statistical data was confirmed by laboratory methods.

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Facilities in the State Board of Health laboratories for the diagnosis of viral disease date back to 1914, when rabies diagnosis began. For many years, this remained the only viral diagnostic test routinely undertaken. Complement fixation test for laboratory diagnosis of typhus was begun in 1944. Other tests for rickettsial and viral diseases were performed as soon as antigens became available. The following serologic tests are now routinely done: Typhus, Rocky Mountain Spotted Fever, Rickettsialpox, "Q" Fever, Eastern and Western Equine Encephalomyelitis, St. Louis Encephalitis, Mumps, Influenza, Psittacosis, Lymphocytic Choriomeningitis, Lymphogranuloma Venereum, and Poliomyelitis (Types I, II and III.)

During 1955, with assistance from the United States Public Health Service, a poliomyelitis virus isolation section was set up in the State Board of Health Laboratory. Tissue culture became a routine laboratory procedure early in 1956.

The outbreak of Asian Influenza, in 1956 and 1957, was responsible for further expansion of the virology laboratory. Isolation of virus, complement fixation, and hemagglutination tests were performed on specimens submitted for diagnosis.

With the aid of the state epidemiologist, a system was set up for collecting stool and blood specimens from as many cases of reported poliomyelitis as can be obtained. Stool specimens are collected as early as possible on three successive days. The first specimen of blood is collected during the acute state, and the second, approximately ten to fourteen days later. The stool specimens are frozen, if possible, and shipped to the laboratory after collection. A laboratory data slip fully filled out accompanies each specimen.

Specimens which show cytopathogenic agents on HeLa cell tissue culture and which do not type with Polio Types I, II and III antisera are sent to the Communicable Disease Center for further study. During an outbreak of supposedly nonparalytic poliomyelitis in the northwestern part of the State in 1957, in which no polio viruses were isolated, 22 cases yielded Echo viruses on monkey kidney cell cultures. Monkey tissue cells, although quite costly and not used routinely in our laboratory, are used when necessary.

Development of Immunization Program

In 1954, the Louisiana State Board of Health participated in the vaccination field trials of the National Foundation for Infantile Paralysis, three parishes being selected, namely, Bossier, Caddo, and Rapides. Their immunization status at the end of 1954, is given in Table 23. It is to be noted that a very high percentage of the children who started the series completed it, receiving all three injections.

From April to September 1955, the NFIP Program was extended to all of the parishes within the State and was offered to all first and second grade children. The following month, the Louisiana State Board of Health embarked on a state-wide polio immunization program, financed by funds specifically appropriated for this purpose by the Congress of the United States. Vaccination was offered to all children 19 years of age and under, and to expectant mothers. For the year of 1955, 59,307 persons had received only one injection of vaccine, 87,113 persons had received two injections, and 2,180 had received three injections. This program was continued without major change through April 1958, at which time the vaccine purchased with federal funds was exhausted. From that time, the program was financed by State appropriated funds. The number of immunizations given for the three-year period 1956-58 is shown in Table 24.

The total polio vaccine injections given from the beginning of the 1954 field trials through April 1958, at which time the federally sponsored program ceased, was 2,362,186, of which 956,173 were first injections, 791,997 were second injections, and 614,016 were third injections.

Beginning in May of 1958, the Louisiana State Board of Health recommended that all persons 40 years of age and un-

der be immunized. Vaccine was offered through local health units to all persons who ordinarily make use of health unit services. During 1958, the State Board of Health's method of tabulating polio injections was changed to conform with the recording methods of other types of immunizations, i.e., considering an individual immunized after 2 injections, the third being a booster.* During 1958, a total of 163,523 persons received two injections of polio vaccine, and, in addition, 246,495 persons received the third, or booster, injection.

Analysis of Statistical Data

In preparing the tables showing the rates of poliomyelitis among the vaccinated and nonvaccinated groups for the years 1956-1958, inclusive, (Tables 25 to 30), several assumptions had to be made. Since practically all of our immunization activities through 1958 had been mainly directed toward individuals under 20 years of age, the total population of individuals 19 years of age and under was used as the basis of these calculations. For the year 1956, the immunized population was calculated by adding together the total number of persons immunized in the field trials of 1954, the total number immunized during the year 1955, and one-half of the total number immunized during 1956. The population in the nonvaccinated group was calculated by taking the total state population for the age-group 19 years and under, and subtracting the total number of immunized persons for the particular year. For 1957 and 1958, this same procedure was used, i.e., all completed immunizations for the prior years plus one-half of the total number immunized in the current year were used to calculate the vaccinated population, and for each year, the total population in the age-group 19 years and under less the vaccinated group gave the total population in the unvaccinated group. It must also be remembered that practically all of these data came mostly from the vaccinations given in the local health units, and take into account only small numbers of children who had been immunized by their family physicians before the vaccine became available through private sources.

During the three year period since the large scale vaccination program began, the paralytic case rates in the vaccinated group decreased 83 per cent below the nonvaccinated group in 1956 (from 43.0 cases per 100,000 nonvaccinated to 7.4 per 100,000 vaccinated persons), 74 per cent below the nonvaccinated in 1957 (from 9.4 to 2.5), and 87 per cent in 1958 (from 10.7 to 1.4). (See Table 25) The vaccinated group included all persons below 20 years of age having had at least 2 injections of vaccine. The rest were considered nonvaccinated. Table 26 shows that similar differences in paralytic case rates were observed between the vaccinated and nonvaccinated, except that the vaccinated group included only those with at least 3 injections.

Tables 27 and 28 make similar comparisons for nonparalytic case rates between vaccinated and nonvaccinated, but in these instances there appear to be no significant differences between the vaccinated and nonvaccinated rates.

Tables 29 and 30 make similar comparisons for rates of all cases of poliomyelitis between the vaccinated and nonvaccinated. Here, the differences are significant, and would be expected to be so because the data include the paralytic cases where differences are great. It can be said, therefore, that the analysis reveals *significant decreases in the paralytic case rates* when comparison is made between the vaccinated and nonvaccinated groups.

If we examine Table 6 for the case rates and death rates of poliomyelitis for the years 1948 to 1958 inclusive, the trend appears to indicate that we are now in the trough of a cycle. This makes it difficult to determine the effectiveness of the vaccine on the basis of these two factors alone. It is, however, worth noting that during the last two years, 1957 and 1958,

* In 1959 this was changed again, considering a person immunzed after three injections, the fourth being a booster.

the death rates were significantly lower than for any years of the study. Could this be attributed to the effectiveness of the vaccine, or to a statistical uncertainty resulting from the small number of cases reported and the very small number of deaths occurring during these two years?

During the years when smaller number of paralytic cases occurred, i.e., 1948, 1957, 1958, the case fatality rates based on paralytic cases were higher than during the other years with larger number of paralytic cases except for the year 1950. The case fatality rate based on all cases was much higher in 1958 than in the other years. It is difficult to explain these findings. We must, however, consider the possibility that small numbers of cases and deaths may cause this variation. But why should the result be only in one direction, i.e., increased fatality rates? Could it be that this one way direction of the case fatality rates based on paralytic cases may be due to the actual decrease in paralytic cases due to the effectiveness of the vaccine? The latter consideration is not confuted by the apparent increased ratio of paralytic to nonparalytic cases in 1958, since this could very well be due to better laboratory and more circumspect clinical diagnosis of nonparalytic cases (Table 6). The possibility of the variable antigenic character of the vaccine for the various strains of poliomyelitis should also be kept in mind.

Several ecological factors which are recognized as important in the epidemiology of communicable diseases are studied here in relation to poliomyelitis death rates, case rates, and paralytic case rates. Although there appears to be some constancy in the ratios between poliomyelitis death rates, case rates, and paralytic case rates for the whole state, year by year from 1949 to 1958 (Tables 5 and 6), this is not so when comparisons are made parish by parish, even when basing the calculation on the summation of cases and deaths for the whole eleven year period (Tables 1, 2, 3, and 4). The reasons for this appear to be, first of all, that except for the larger cities, the number of cases and deaths are too limited to be as statistically valid as would be desirable, and secondly, that the problems of satisfactory diagnosis have not been settled. The death rates, therefore, are the most valid statistics for poliomyelitis, and paralytic cases are second. But nonparalytic cases must be considered as data that most probably are too variable for satisfactory statistical study. Case rates have, nevertheless, been used in this study of correlation.

In order to derive epidemiological information in relation to various ecological factors, the statistical technique used in this study is the same as previously used for pinpointing Louisiana health problems.[1] The following interrelationships were derived:

1. There appears to be some indication of correlation between higher average poliomyelitis death rates and lower population densities in those parishes having average poliomyelitis death rates higher than 1.0 per 100,000 population, i.e., the lower the population densities in such parishes, the more likely are their average death rates expected to be higher in the Louisiana experience (Table 7). One would expect that the immunity status in less dense areas would be less, thereby predisposing to higher death rates.

2. There appears to be some correlation between average poliomyelitis case rates and lower population densities in those parishes having average poliomyelitis case rates lower than 15 cases per 100,000 population, i.e., the lower the population densities in such parishes, the more likely, in the Louisiana experience, are their average poliomyelitis case rates expected to be lower (Table 8). This could be due to the retarded force of the crowding factor. It might conceivably be due also to the lesser medical services

[1] B. Freedman: Pinpointing Public Health Needs in Louisiana, Journal La. State Medical Soc., 110:71-92 (March) 1958.

(Continued on page 358)

TABLE 1

POLIOMYELITIS AVERAGE DEATH RATES, CASE RATES, AND PARALYTIC CASE RATES PER 100,000 POPULATION, 1948-58, RELATED TO THE POPULATION DENSITY PER SQUARE MILE, 1955, IN EACH PARISH IN LOUISIANA

Parishes Listed According to their Population Density per square mile from Lowest to Highest Densities	Density per sq. mile 1955	Poliomyelitis Average Rates for 1948-1958 Period		
		Death Rates	Case Rates	Paralytic Case Rates
		See Table 7	See Table 8	See Table 9
01. Cameron	4.0	3.1	9.9	6.4
02. Vernon	13.9	1.6	44.2	24.4
03. Catahoula	14.2	3.4	16.5	10.0
04. Plaquemines	15.5	0.0	6.6	6.6
05. Beauregard	16.3	1.0	8.5	4.3
06. Winn	16.5	1.2	9.8	7.3
07. Caldwell	17.0	0.0	2.1	2.1
08. Sabine	18.9	1.0	17.2	12.5
09. Tensas	18.9	0.0	7.4	6.0
10. Grant	20.0	0.0	9.7	7.0
11. Bienville	20.1	0.0	16.0	9.7
12. Union	20.1	1.1	8.2	6.2
13. Concordia	20.2	2.7	8.0	5.4
14. St. Helena	20.8	0.0	11.1	8.9
15. LaSalle	21.4	1.5	12.9	10.1
16. W. Feliciana	22.8	1.7	11.3	3.5
17. DeSoto	23.0	0.8	18.6	10.9
18. Jackson	24.3	1.3	10.3	7.6
19. Red River	24.5	3.5	32.2	26.8
20. Allen	25.2	1.1	12.0	9.0
21. Madison	25.6	1.2	11.2	5.6
22. St. Bernard	25.7	0.0	20.8	17.6
23. Natchitoches	28.3	0.9	12.3	8.1
24. Claiborne	29.4	0.8	20.3	12.2
25. Vermilion	29.8	0.5	13.8	9.0
26. St. Tammany	31.7	1.4	15.8	13.0
27. Livingston	31.9	1.0	25.2	19.3
28. Terrebonne	34.0	0.4	12.0	9.7
29. E. Carroll	34.4	1.2	14.7	7.3
30. St. Martin	36.5	0.0	4.4	4.4
31. Pointe Coupee	36.7	0.9	18.0	13.6
32. Lafourche	38.1	0.5	19.5	12.3
33. Jefferson Davis	41.7	0.0	10.7	5.8
34. Iberville	41.8	1.5	11.0	8.0
35. Morehouse	42.8	2.4	20.1	13.2
36. Franklin	42.9	0.0	5.1	3.9
37. E. Feliciana	43.4	0.0	11.4	10.4
38. Richland	44.3	1.5	38.8	15.4
39. Avoyelles	45.3	0.0	23.0	10.7
40. W. Carroll	45.5	0.0	25.3	9.2
41. St. Charles	45.8	0.0	13.3	7.4
42. Assumption	46.5	0.0	12.6	5.6
43. Evangeline	47.9	1.2	5.0	5.0
44. Bossier	52.1	1.0	32.7	18.3
45. Lincoln	56.1	0.8	14.6	8.5
46. Webster	58.7	1.1	18.9	12.9
47. St. James	58.9	0.0	7.6	6.4
48. W. Baton Rouge	59.7	3.2	16.3	11.4
49. Washington	60.8	0.5	10.1	8.5
50. St. Mary	63.1	1.6	12.6	11.0
51. St. John	66.3	0.0	10.4	9.1
52. Iberia	70.7	0.0	6.2	5.7
53. Tangipahoa	71.3	0.4	11.1	9.5
54. Acadia	71.7	0.8	9.0	5.7
55. Rapides	75.0	1.1	20.5	9.5
56. Ascension	76.7	0.9	12.8	10.3
57. St. Landry	88.3	0.7	10.7	8.3
58. Calcasieu	96.9	0.6	14.0	10.7
59. Ouachita	129.1	0.8	25.5	14.9
60. Caddo	213.7	1.1	36.4	22.9
61. Lafayette	229.6	1.3	16.1	10.3
62. Jefferson	322.6	0.3	13.3	10.7
63. E. Baton Rouge	421.8	0.6	30.9	18.3
64. Orleans	3066.8	0.4	8.2	6.5

TABLE 2

POLIOMYELITIS AVERAGE DEATH RATES, CASE RATES, AND PARALYTIC CASE RATES PER 100,000 POPULATION, 1948-58, RELATED TO THE RURALITY, 1950, OF EACH PARISH IN LOUISIANA

Parishes Listed According to their Rurality, from the Highest % to the Lowest % Rural Population	% Rural Population 1950	Poliomyelitis Average Case Rates 1948-1958 Period		
		Death Rates	Case Rates	Paralytic Case Rates
		See Table 10	See Table 11	See Table 12
01. Cameron	100.0	3.1	9.9	6.4
02. Catahoula	100.0	3.4	16.5	10.0
03. Plaquemines	100.0	0.0	6.6	6.6
04. Caldwell	100.0	0.0	2.1	2.1
05. Sabine	100.0	1.0	17.2	12.5
06. Tensas	100.0	0.0	7.4	6.0
07. Grant	100.0	0.0	9.7	7.0
08. Bienville	100.0	0.0	16.0	9.7
09. Union	100.0	1.1	8.2	6.2
10. St. Helena	100.0	0.0	11.1	8.9
11. LaSalle	100.0	1.5	12.9	10.1
12. W. Feliciana	100.0	1.7	11.3	3.5
13. Red River	100.0	3.5	32.2	26.8
14. Livingston	100.0	1.0	25.2	19.2
15. W. Carroll	100.0	0.0	25.3	9.2
16. Assumption	100.0	0.0	12.6	5.6
17. St. James	100.0	0.0	7.6	6.4
18. Richland	88.3	1.5	38.8	15.4
19. Franklin	87.6	0.0	5.1	3.9
20. Pointe Coupee	87.1	0.9	18.0	13.6
21. St. Martin	82.5	0.0	4.4	4.4
22. DeSoto	81.8	0.8	18.6	10.9
23. Ascension	81.5	0.9	12.8	10.3
24. Jackson	79.9	1.3	10.3	7.6
25. Evangeline	79.0	1.2	5.0	5.0
26. Iberville	78.6	1.5	11.0	8.0
27. Avoyelles	78.2	0.0	23.0	10.7
28. St. Bernard	76.2	0.0	20.8	17.6
29. Vernon	75.4	1.6	44.2	24.4
30. Lafourche	75.0	0.5	19.5	12.3
31. St. Charles	74.9	0.0	13.3	7.4
32. St. Landry	74.7	0.7	10.7	8.3
33. E. Carroll	74.7	1.2	14.7	7.3
34. Natchitoches	74.0	0.9	12.3	8.1
35. W. Baton Rouge	73.6	3.2	16.3	11.4
36. Concordia	73.3	2.7	8.0	5.4
37. Tangipahoa	72.0	0.4	11.1	9.5
38. Allen	70.3	1.1	12.0	9.0
39. St. John	70.0	0.0	10.4	9.1
40. Claiborne	68.9	0.8	20.3	12.2
41. St. Tammany	68.2	1.4	15.8	13.0
42. Beauregard	67.4	1.0	8.5	4.3
43. Winn	65.0	1.2	9.8	7.3
44. E. Feliciana	64.6	0.0	11.4	10.4
45. Webster	63.2	1.1	18.9	12.9
46. Terrebonne	62.3	0.4	12.0	9.7
47. Vermilion	62.3	0.5	13.8	9.0
48. Morehouse	60.1	2.4	20.1	13.2
49. Lincoln	59.8	0.8	14.6	8.5
50. Bossier	58.2	1.0	32.7	18.3
51. Madison	55.5	1.2	11.2	5.6
52. Rapides	54.4	1.1	20.5	9.5
53. Washington	53.6	0.5	10.1	8.5
54. Acadia	52.9	0.8	9.0	5.7
55. Jefferson Davis	52.4	0.0	10.7	5.8
56. St. Mary	48.4	1.6	12.6	11.0
57. Iberia	47.1	0.0	6.2	5.7
58. Lafayette	42.0	1.3	16.1	10.3
59. Ouachita	34.6	0.8	25.5	14.9
60. Calcasieu	27.8	0.6	14.0	10.7
61. Caddo	24.4	1.1	36.4	22.9
62. E. Baton Rouge	14.2	0.6	30.9	18.3
63. Jefferson	11.2	0.3	13.3	10.7
64. Orleans	0.0	0.4	8.2	6.5

TABLE 3

POLIOMYELITIS AVERAGE DEATH RATES, CASE RATES, AND PARALYTIC CASE RATES PER 100,000 POPULATION, 1948-58, RELATED TO THE MEDIAN ANNUAL INCOME OF FAMILIES AND UNRELATED INDIVIDUALS, 1950, IN EACH PARISH IN LOUISIANA

Parishes Listed According to their Median Annual Income for Families and Unrelated Individuals from Lowest to Highest	Median Annual Income	Poliomyelitis Average Rates for 1948-1958 Period		
		Death Rates	Case Rates	Paralytic Case Rates
		See Table 13	See Table 14	See Table 15
01. E. Carroll	$ 835	1.2	14.7	7.3
02. Tensas	840	0.0	7.4	6.0
03. Natchitoches	965	0.9	12.3	8.1
04. Madison	982	1.2	11.2	5.6
05. E. Feliciana	1,007	0.0	11.4	10.4
06. Red River	1,030	3.5	32.2	26.8
07. Concordia	1,058	2.7	8.0	5.4
08. St. Helena	1,058	0.0	11.1	8.9
09. W. Carroll	1,064	0.0	25.3	9.2
10. Vernon	1,089	1.6	44.2	24.4
11. W. Feliciana	1,100	1.7	11.3	3.5
12. Catahoula	1,109	3.4	16.5	10.0
13. St. James	1,118	0.0	7.6	6.4
14. Pointe Coupee	1,124	0.9	18.0	13.6
15. DeSoto	1,164	0.8	18.6	10.9
16. Assumption	1,169	0.0	12.6	5.6
17. Grant	1,194	0.0	9.7	7.0
18. Tangipahoa	1,204	0.4	11.1	9.5
19. Lincoln	1,212	0.8	14.6	8.5
20. Sabine	1,214	1.0	17.2	12.5
21. Avoyelles	1,215	0.0	23.0	10.7
22. Richland	1,224	1.5	38.8	15.4
23. Franklin	1,224	0.0	5.1	3.9
24. St. Landry	1,242	0.7	10.7	8.3
25. Evangeline	1,247	1.2	5.0	5.0
26. Vermilion	1,269	0.5	13.8	9.0
27. Bienville	1,272	0.0	16.0	9.7
28. Winn	1,315	1.2	9.8	7.3
29. St. Martin	1,316	0.0	4.4	4.4
30. Caldwell	1,355	0.0	2.1	2.1
31. Iberville	1,381	1.5	11.0	8.0
32. Ascension	1,410	0.9	12.8	10.3
33. W. Baton Rouge	1,437	3.2	16.3	11.4
34. St. Tammany	1,446	1.4	15.8	13.0
35. Claiborne	1,458	0.8	20.3	12.2
36. Union	1,473	1.1	8.2	6.2
37. Morehouse	1,533	2.4	20.1	13.2
38. Acadia	1,563	0.8	4.0	5.7
39. Beauregard	1,573	1.0	8.5	4.3
40. LaSalle	1,613	1.5	12.9	10.1
41. Allen	1,634	1.1	12.0	9.0
42. Livingston	1,645	1.0	25.2	19.3
43. Jefferson Davis	1,656	0.0	10.7	5.8
44. Washington	1,679	0.5	10.1	8.5
45. Lafayette	1,680	1.3	16.1	10.3
46. Rapides	1,690	1.1	20.5	9.5
47. St. John	1,707	0.0	10.4	9.1
48. Bossier	1,744	1.0	32.7	18.3
49. St. Mary	1,831	1.6	12.6	11.0
50. Iberia	1,835	0.0	6.2	5.7
51. Jackson	1,876	1.3	10.3	7.6
52. Terrebonne	1,901	0.4	12.0	9.7
53. St. Charles	1,907	0.0	13.3	7.4
54. Webster	1,920	1.1	18.9	12.9
55. Lafourche	1,920	0.5	19.5	12.3
56. St. Bernard	2,020	0.0	20.8	17.6
57. Ouachita	2,052	0.8	25.5	14.9
58. Cameron	2,090	3.1	9.9	6.4
59. Caddo	2,157	1.1	36.4	22.9
60. Plaquemines	2,197	0.0	6.6	6.6
61. Orleans	2,267	0.4	8.2	6.5
62. E. Baton Rouge	2,618	0.6	30.9	18.3
63. Calcasieu	2,668	0.6	14.0	10.7
64. Jefferson	2,762	0.3	13.3	10.7

TABLE 4

POLIOMYELITIS AVERAGE DEATH RATES, CASE RATES, AND PARALYTIC CASE RATES PER 100,000 POPULATION, 1948-58, RELATED TO THE HOUSING SANITATION, 1950, IN EACH PARISH IN LOUISIANA

Parishes Listed According to their Housing Sanitation from the Worst to the Best	% of Housing with Private Toilets, Baths, and Hot or Cold Running Water 1950	Poliomyelitis Average Case Rates 1948-1958 Period		
		Death Rates	Case Rates	Paralytic Case Rates
		See Table 16	See Table 17	See Table 18
01. West Carroll	12.2	0.0	25.3	9.2
02. Red River	12.5	3.5	32.2	26.8
03. St. Helena	12.7	0.0	11.1	8.9
04. Catahoula	12.8	3.4	16.5	10.0
05. E. Carroll	13.3	1.2	14.7	7.3
06. Grant	13.8	0.0	9.7	7.0
07. Franklin	14.9	0.0	5.1	3.9
08. Tensas	15.1	0.0	7.4	6.0
09. Sabine	15.2	1.0	17.2	12.5
10. Madison	15.3	1.2	11.2	5.6
11. Caldwell	18.4	0.0	2.1	2.1
12. Bienville	18.6	0.0	16.0	9.7
13. W. Feliciana	18.7	1.7	11.3	3.5
14. Union	19.1	1.1	8.2	6.2
15. Natchitoches	19.5	0.9	12.3	8.1
16. Concordia	19.6	2.7	8.0	5.4
17. E. Feliciana	19.8	0.0	11.4	10.4
18. Richland	20.0	1.5	38.8	15.4
19. Assumption	20.0	0.0	12.6	5.6
20. Winn	21.4	1.2	9.8	7.3
21. Pointe Coupee	22.1	0.9	18.0	13.6
22. St. Martin	22.5	0.0	4.4	4.4
23. St. James	22.9	0.0	7.6	6.4
24. Avoyelles	23.5	0.0	23.0	10.7
25. DeSoto	23.7	0.8	18.6	10.9
26. St. John	25.0	0.0	10.4	9.1
27. Vernon	25.4	1.6	44.2	24.4
28. Evangeline	25.5	1.2	5.0	5.0
29. Morehouse	26.5	2.4	20.1	13.2
30. St. Landry	26.7	0.7	10.7	8.3
31. Cameron	27.4	3.1	9.9	6.4
32. LaSalle	27.8	1.5	12.9	10.1
33. Livingston	27.9	1.0	25.2	19.3
34. Iberville	28.3	1.5	11.0	8.0
35. Allen	29.7	1.1	12.0	9.0
36. Claiborne	30.0	0.8	20.3	12.2
37. W. Baton Rouge	30.1	3.2	16.3	11.4
38. Ascension	30.6	0.9	12.8	10.3
39. Jackson	31.6	1.3	10.3	7.6
40. Tangipahoa	32.4	0.4	11.1	9.5
41. Vermilion	33.2	0.5	13.8	9.0
42. Beauregard	34.0	1.0	8.5	4.3
43. Acadia	35.0	0.8	9.0	5.7
44. Plaquemines	35.9	0.0	6.6	6.6
45. Lincoln	36.2	0.8	14.6	8.5
46. Lafourche	37.2	0.5	19.5	12.3
47. Jefferson Davis	37.3	0.0	10.7	5.8
48. St. Charles	38.1	0.0	13.3	7.4
49. St. Mary	38.3	1.6	12.6	11.0
50. Washington	38.9	0.5	10.1	8.5
51. Terrebonne	39.4	0.4	12.0	9.7
52. Webster	42.8	1.1	18.9	12.9
53. Rapides	43.7	1.1	20.5	9.5
54. Bossier	44.1	1.0	32.7	18.3
55. Ouachita	45.2	0.8	25.5	14.9
56. Iberia	46.0	0.0	6.2	5.7
57. St. Tammany	49.4	1.4	15.8	13.0
58. Lafayette	52.5	1.3	16.1	10.3
59. St. Bernard	52.6	0.0	20.8	17.6
60. Caddo	54.5	1.1	36.4	22.9
61. Calcasieu	55.2	0.6	14.0	10.7
62. Jefferson	68.4	0.3	13.3	10.7
63. E. Baton Rouge	70.0	0.6	30.9	18.3
64. Orleans	74.7	0.4	8.2	6.5

TABLE 5
POLIOMYELITIS CASES AND DEATHS, LOUISIANA
1948-1958

Year	Total Polio Cases	Number of Paralytic Polio Cases	Number of Non-Paralytic Polio Cases	Number of Polio Deaths
1948	163	163	------	19
1949	227	227	------	10
1950	409	278	131	22
1951	867	578	289	36
1952	871	613	258	34
1953	438	209	229	12
1954	537	319	218	16
1955	383	242	141	13
1956	608	414	194	23
1957	169	74	95	6
1958	75	54	21	5

TABLE 6
POLIOMYELITIS RATES AND RATIOS, LOUISIANA
1948-1958

Year	Polio Death Rates per 1,000 Cases of Polio	Polio Death Rates per 1,000 Paralytic Cases	Ratio of all Polio Cases to Paralytic Cases	Ratio of Paralytic to Non-Paralytic Cases	Polio Case Rates per 100,000 Population	Polio Death Rates per 100,000 Population
1948		116.5				0.72
1949		44.0				0.38
1950	53.7	79.0	1.5:1	2:1	15.34	0.82
1951	41.5	62.0	1.5:1	2:1	31.83	1.32
1952	39.0	55.0	1.4:1	2.4:1	31.47	1.23
1953	27.0	57.0	2.1:1	0.9:1	15.71	0.43
1954	29.7	50.0	1.7:1	1.5:1	19.05	0.57
1955	33.9	53.7	1.6:1	1.7:1	13.43	0.46
1956	37.8	55.5	1.5:1	2:1	21.09	0.80
1957	35.5	81.0	2.2:1	0.8:1	5.40	0.19
1958	66.7	92.6	1.4:1	2.5:1	2.35	0.16

TABLE 7
PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR POPULATION DENSITY PER SQUARE MILE, AND GROUPED ACCORDING TO THEIR AVERAGE DEATH RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Death Rates per 100,000 Population in each Parish with a greater rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to Highest Population Densities per square mile			
	1st Quartile (Parishes with Lowest Population Densities)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Population Densities)
3.0	2 parishes	1 parish	1 parish	0 parish
2.5	3 parishes	1 parish	1 parish	0 parish
2.0	3 parishes	1 parish	2 parishes	0 parish
1.5	5 parishes	1 parish	2 parishes	1 parish
1.0	8 parishes	6 parishes	6 parishes	4 parishes
0.5	10 parishes	11 parishes	8 parishes	10 parishes

There appears to be some indication of correlation between higher average poliomyelitis death rates and lower population densities in those parishes having average poliomyelitis death rates higher than 1.0 per 100,000 population, i.e., the lower the population densities in such parishes, the more likely, in the Louisiana experience, are their average poliomyelitis death rates expected to be higher.

TABLE 8

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR POPULATION DENSITY PER SQUARE MILE, AND GROUPED ACCORDING TO THEIR AVERAGE POLIOMYELITIS CASE RATES PER 100,000 POPULATION FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to Highest Population Densities per square mile			
	1st Quartile (Parishes with Lowest Population Densities)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Population Densities)
10	9 parishes	1 parish	3 parishes	3 parishes
15	12 parishes	8 parishes	9 parishes	11 parishes
20	15 parishes	12 parishes	11 parishes	12 parishes
25	15 parishes	14 parishes	13 parishes	13 parishes
30	15 parishes	15 parishes	14 parishes	14 parishes

There appears to be some correlation between lower average poliomyelitis case rates and lower population densities in those parishes having average poliomyelitis case rates lower than 15 cases per 100,000 population, i.e., the lower the population densities in such parishes, the more likely, in the Louisiana experience, are their average poliomyelitis case rates expected to be lower.

TABLE 9

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR POPULATION DENSITY PER SQUARE MILE, AND GROUPED ACCORDING TO THEIR AVERAGE POLIOMYELITIS PARALYTIC CASE RATES PER 100,000 POPULATION FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Paralytic Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to the Highest Population Densities per square mile			
	1st Quartile (Parishes with Lowest Population Densities)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Population Densities)
5.0	3 parishes	1 parish	1 parish	0 parish
7.5	10 parishes	3 parishes	6 parishes	3 parishes
10.0	12 parishes	8 parishes	9 parishes	8 parishes
12.5	14 parishes	11 parishes	12 parishes	13 parishes
15.0	15 parishes	13 parishes	14 parishes	14 parishes

There appears to be fairly evident correlation between lower average poliomyelitis paralytic case rates and lower population densities in those parishes having average paralytic case rates less than 10 cases per 100,000 population, i.e., the lower the population densities in such parishes, the more likely, in the Louisiana experience, are their average poliomyelitis paralytic case rates expected to be lower.

TABLE 10

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF RURALITY, AND GROUPED ACCORDING TO THEIR AVERAGE DEATH RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Death Rates per 100,000 Population in each Parish with a greater rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Highest Degree of Rurality to the Lowest			
	1st Quartile (Parishes with Highest Degree of Rurality)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Lowest Degree of Rurality)
3.0	3 parishes	0 parish	1 parish	0 parish
2.5	3 parishes	0 parish	2 parishes	0 parish
2.0	3 parishes	0 parish	3 parishes	0 parish
1.5	4 parishes	1 parish	3 parishes	1 parish
1.0	6 parishes	5 parishes	8 parishes	5 parishes
0.5	8 parishes	9 parishes	11 parishes	11 parishes

There appears to be no correlation between the average poliomyelitis death rates of parishes and their degree of rurality.

TABLE 11

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF RURALITY, AND GROUPED ACCORDING TO THEIR AVERAGE CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Highest Degree of Rurality to the Lowest			
	1st Quartile (Parishes with Highest Degree of Rurality)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Lowest Degree of Rurality)
10	6 parishes	4 parishes	3 parishes	3 parishes
15	10 parishes	9 parishes	10 parishes	10 parishes
20	13 parishes	12 parishes	14 parishes	11 parishes
25	13 parishes	14 parishes	16 parishes	12 parishes
30	15 parishes	14 parishes	16 parishes	13 parishes

There appears to be no correlation between the average poliomyelitis case rates of parishes and their degree of rurality.

TABLE 12

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF RURALITY, AND GROUPED ACCORDING TO THEIR AVERAGE PARALYTIC CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Paralytic Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Highest Degree of Rurality to the Lowest			
	1st Quartile (Parishes with Highest Degree of Rurality)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Lowest Degree of Rurality)
5.0	2 parishes	2 parishes	1 parish	0 parish
7.0	8 parishes	5 parishes	4 parishes	5 parishes
10.0	11 parishes	8 parishes	10 parishes	8 parishes
12.5	13 parishes	12 parishes	13 parishes	12 parishes
15.0	14 parishes	13 parishes	16 parishes	13 parishes

Although there appears to be a slight tendency of correlation between higher degree of rurality and lower average poliomyelitis paralytic case rates in those parishes having paralytic case rates less than 10 per 100,000 population, such a relationship can really not be deduced since even the slight tendency is scarcely enough to claim even slight validity.

TABLE 13

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR MEDIAN ANNUAL INCOME IN 1950, AND GROUPED ACCORDING TO THEIR AVERAGE DEATH RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Death Rates per 100,000 Population in each Parish with a greater rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to Highest Median Annual Income			
	1st Quartile (Parishes with Lowest Median Annual Income)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Median Annual Income)
3.0	1 parish	0 parish	2 parishes	1 parish
2.5	2 parishes	0 parish	2 parishes	1 parish
2.0	3 parishes	0 parish	2 parishes	1 parish
1.5	4 parishes	2 parishes	2 parishes	1 parish
1.0	8 parishes	7 parishes	5 parishes	4 parishes
0.5	13 parishes	10 parishes	9 parishes	7 parishes

There appears to be some indication of correlation between higher average poliomyelitis death rates and lower median annual income of families, i.e., the lower the median annual income of families in parishes, the more likely, in the Louisiana experience, are the average poliomyelitis death rates expected to be higher in such parishes.

TABLE 14

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR MEDIAN ANNUAL INCOME IN 1950, AND GROUPED ACCORDING TO THEIR AVERAGE CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to Highest Median Annual Income			
	1st Quartile (Parishes with Lowest Median Annual Income)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Median Annual Income)
10	3 parishes	6 parishes	3 parishes	4 parishes
15	10 parishes	12 parishes	8 parishes	10 parishes
20	13 parishes	14 parishes	11 parishes	12 parishes
25	13 parishes	15 parishes	14 parishes	13 parishes
30	14 parishes	15 parishes	15 parishes	14 parishes

There appears to be no correlation between the average poliomyelitis case rates of Parishes and their median annual income of families.

TABLE 15

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR MEDIAN ANNUAL INCOME IN 1950, AND GROUPED ACCORDING TO THEIR AVERAGE PARALYTIC CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Paralytic Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Lowest to the Highest Median Annual Income			
	1st Quartile (Parishes with Lowest Median Annual Income)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Median Annual Income)
5.0	1 parish	3 parishes	1 parish	0 parish
7.5	7 parishes	6 parishes	4 parishes	5 parishes
10.0	10 parishes	12 parishes	8 parishes	7 parishes
12.5	13 parishes	14 parishes	12 parishes	11 parishes
15.0	14 parishes	15 parishes	14 parishes	13 parishes

There appears to be no correlation between the average poliomyelitis paralytic case rates of parishes and their median annual income of families.

TABLE 16

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF HOUSING SANITATION, AND GROUPED ACCORDING TO THEIR AVERAGE DEATH RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Death Rates per 100,000 Population in each Parish with a greater rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Lowest to the Highest Degree of Housing Sanitation			
	1st Quartile (Parishes with Lowest Degree of Housing Sanitation)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Degree of Housing Sanitation)
3.0	2 parishes	1 parish	1 parish	0 parish
2.5	3 parishes	1 parish	1 parish	0 parish
2.0	3 parishes	2 parishes	1 parish	0 parish
1.5	4 parishes	3 parishes	1 parish	1 parish
1.0	7 parishes	7 parishes	4 parishes	6 parishes
0.5	9 parishes	10 parishes	10 parishes	10 parishes

There appears to be no evidence of correlation between average poliomyelitis death rates and the degree of housing sanitation.

TABLE 17

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF HOUSING SANITATION, AND GROUPED ACCORDING TO THEIR AVERAGE CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD 1948-1958

Average Poliomyelitis Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from the Lowest to the Highest Degree of Housing Sanitation			
	1st Quartile (Parishes with Lowest Degree of Housing Sanitation)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Degree of Housing Sanitation)
10	6 parishes	5 parishes	3 parishes	2 parishes
15	11 parishes	10 parishes	12 parishes	7 parishes
20	14 parishes	12 parishes	14 parishes	10 parishes
25	14 parishes	14 parishes	15 parishes	12 parishes
30	15 parishes	14 parishes	16 parishes	13 parishes

There appears to be no evidence of correlation between average poliomyelitis case rates and degree of housing sanitation.

TABLE 18

PARISHES IN LOUISIANA CLASSIFIED INTO QUARTILES ACCORDING TO THEIR DEGREE OF HOUSING SANITATION, AND GROUPED ACCORDING TO THEIR AVERAGE PARALYTIC CASE RATES PER 100,000 POPULATION FROM POLIOMYELITIS FOR THE 11 YEAR PERIOD, 1948-1958

Average Poliomyelitis Paralytic Case Rates per 100,000 Population in each Parish with a lower rate than	Parishes arranged into Quartiles (16 parishes in each Quartile) from Lowest to Highest Degree of Housing Sanitation			
	1st Quartile (Parishes with Lowest Degree of Housing Sanitation)	2nd Quartile	3rd Quartile	4th Quartile (Parishes with Highest Degree of Housing Sanitation)
5.0	3 parishes	1 parish	1 parish	0 parish
7.5	9 parishes	6 parishes	5 parishes	2 parishes
10.0	13 parishes	8 parishes	11 parishes	5 parishes
12.5	14 parishes	12 parishes	15 parishes	9 parishes
15.0	15 parishes	14 parishes	15 parishes	12 parishes

There appears to be evidence of correlation between the average poliomyelitis paralytic case rates and housing sanitation, i.e., the poorer the housing sanitation of parishes, the more likely, in the Louisiana experience, are more of these parishes expected to show lower paralytic case rates.

TABLE 19

LABORATORY FINDINGS BY VIRUS ISOLATION ON 406 CASES OF CLINICALLY DIAGNOSED POLIOMYELITIS, AND VACCINATION STATUS OF CASES, LOUISIANA, 1956

Clinical Diagnosis and Vaccination Status	Virus Types Isolated*						Total Isolations	No Isolations	Totals
	I	II	III	I & III	Coxsackie	Echo			
Paralytic Cases	137	3	40	1	0	0	181	55	236
Vaccination Status									
1 injection	15	0	3	0	0	0			
2 injections	3	0	1	0	0	0			
3 injections	0	0	0	0	0	0			
None	119	3	36	1	0	0			
Non-Paralytic Cases	43	1	7	0	1	0	52	118	170
Vaccination Status									
1 injection	3	0	1	0	0	0			
2 injections	0	0	0	0	0	0			
3 injections	2	0	0	0	0	0			
None	38	1	6	0	1	0			
TOTALS	180	4	47	1	1	0	233	173	406

* No complement-fixation tests were made.

TABLE 20

LABORATORY FINDINGS BY VIRUS ISOLATION ON 125 CASES OF CLINICALLY DIAGNOSED POLIOMYELITIS, AND VACCINATION STATUS OF CASES, LOUISIANA, 1957

Clinical Diagnosis and Vaccination Status	Virus Types Isolated*					Total Isolations	No Isolations	Totals
	I	II	III	Coxsackie	Echo			
Paralytic Cases	4	1	1	0	1	7	29	36
Vaccination Status								
1 injection	1	0	unknown	0	0			
2 injections	1	0	unknown	0	0			
3 injections	0	0	unknown	0	0			
None	2	1	unknown	0	1			
Non-Paralytic Cases	5	0	0	4	18	27	62	89
Vaccination Status								
1 injection	0	0	0	1	0			
2 injections	0	0	0	0	2			
3 injections	0	0	0	0	4			
None	5	0	0	3	12			
TOTALS	9	1	1	4	19	34	91	125

* No complement-fixation tests were made.

TABLE 21

LABORATORY FINDINGS BY VIRUS ISOLATION ON 87 CASES OF CLINICALLY DIAGNOSED POLIOMYELITIS, AND VACCINATION STATUS OF CASES, LOUISIANA, 1958

Clinical Diagnosis and Vaccination Status	Virus Types Isolated					Total Isolations	No Isolations	Totals
	I	II	III	Coxsackie	Echo			
Paralytic Cases	9	0	9	0	1	19	13	32
Vaccination Status								
1 injection	1	0	1	0	0			
2 injections	0	0	1	0	0			
3 injections	1	0	1	0	0			
None	7	0	6	0	1			
Non-Paralytic Cases	2	0	0	2	0	4	51	55
Vaccination Status								
1 injection	1	0	0	0	0			
2 injections	0	0	0	0	0			
3 injections	0	0	0	2	0			
None	1	0	0	0	0			
TOTALS	11	0	9	2	1	23	64	87

TABLE 22

RESULTS OF COMPLEMENT FIXATION TESTS DONE ON 87 CASES OF CLINICALLY DIAGNOSED POLIOMYELITIS (SAME CASES AS STUDIED IN TABLE 21) LOUISIANA, 1958

Clinical Diagnosis	Positives, Types			Total Positives	Inconclusive Results	Negatives	Unsatisfactory Specimens	Totals
	I	II	III					
Paralytic	7	0	7	14	1	8	9	32
Non-Paralytic	2	0	2	4	1	22	28	55

TABLE 23
POLIO VACCINE INJECTIONS IN THREE LOUISIANA PARISHES
1954

Parish	First Injection	Second Injection	Third Injection
Bossier	542	542	537
Caddo	2509	2480	2383
Rapides	735	718	702
TOTAL	3786	3740	3622

TABLE 24
POLIOMYELITIS VACCINE INJECTIONS
LOUISIANA
1956-1958

Year	First Injection	Second Injection	Third Injection
1956	333,273	302,116	150,144
1957	374,517	326,443	298,941
1958*	95,997	70,405	159,129
Totals	803,787	698,964	608,214

* From January through April only, at which time the federally sponsored program was discontinued.

TABLE 25
PARALYTIC POLIOMYELITIS RATES PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 2 Injections Considered as Vaccinated)
LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Paralytic Cases	Rate	Population	Paralytic Cases	Rate
1956	244,091	18	7.4	916,380	394	43.0
1957	558,370	14	2.5	637,101	60	9.4
1958	803,353	11	1.4	427,118	43	10.7

TABLE 26
PARALYTIC POLIOMYELITIS RATES PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 3 Injections Considered as Vaccinated)
LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Paralytic Cases	Rate	Population	Paralytic Cases	Rate
1956	80,874	0	0	1,079,597	412	38.1
1957	305,416	5	1.6	890,055	69	7.7
1958	578,134	6	1.0	652,337	48	7.3

TABLE 27

NONPARALYTIC POLIOMYELITIS RATES PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 2 Injections Considered as Vaccinated)

LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Non-Paralytic Cases	Rate	Population	Non-Paralytic Cases	Rate
1956	244,091	27	11.1	916,380	167	18.2
1957	558,370	41	7.3	637,101	54	8.5
1958	803,353	13	1.6	427,118	8	1.9

TABLE 28

NONPARALYTIC POLIOMYELITIS RATES PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 3 Injections Considered as Vaccinated)

LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Non-Paralytic Cases	Rate	Population	Non-Paralytic Cases	Rate
1956	80,874	7	8.6	1,079,597	187	17.3
1957	305,416	22	7.2	890,055	73	8.2
1958	578,134	11	1.9	652,337	10	1.5

TABLE 29

POLIOMYELITIS RATES (TOTAL CASES) PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 2 Injections Considered as Vaccinated)

LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Total Cases	Rate	Population	Total Cases	Rate
1956	244,091	45	18.4	916,380	561	61.2
1957	558,370	55	9.9	637,101	114	17.9
1958	803,353	24	3.0	427,118	51	11.9

TABLE 30

POLIOMYELITIS RATES (TOTAL CASES) PER 100,000 PERSONS IN VACCINATED AND NON-VACCINATED GROUPS OF THE POPULATION UNDER 20 YEARS OF AGE
(On Basis of 3 Injections Considered as Vaccinated)

LOUISIANA
1956-1958

	Vaccinated			Non-Vaccinated		
Year	Population	Total Cases	Rate	Population	Total Cases	Rate
1956	80,874	7	8.6	1,079,597	599	55.4
1957	305,416	27	8.8	890,055	142	16.0
1958	578,134	17	2.9	652,337	58	8.8

available in the more sparsely populated areas, and, therefore, less number of medically diagnosed cases.

3. There appears to be fairly evident correlation between lower average poliomyelitis paralytic case rates and lower population densities in those parishes having average paralytic case rates less than 10 cases per 100,000 population, i.e., the lower the population densities in such parishes, the more likely, in the Louisiana experience, are their average poliomyelitis paralytic case rates expected to be lower (Table 9). The ratio of poliomyelitis case rates to paralytic rates has remained fairly constant in the last decade (Table 6), therefore, this table (No. 9) should show similar interrelationships as in Table 8, which, of course, it does.

4. The degree of rurality of the parishes does not show any correlation with average poliomyelitis death rates, case rates or paralytic case rates. (Tables 10, 11, and 12.) The nature of the definition of rurality usually makes it a poorer, less stable factor to use than population density when considering the epidemiology of communicable diseases.

5. There appears to be some indication of correlation between higher average poliomyelitis death rates and lower median annual income of families, i.e., the lower the median annual income of families the more likely, in the Louisiana experience, are the average poliomyelitis death rates expected to be higher in such parishes; but no correlation was found between either the average poliomyelitis case rates or paralytic case rates and the median annual income of families (Tables 13, 14, and 15).

Decreased income and increased death rates usually correlate for most diseases. The reporting of deaths is much more accurate than reporting of morbidity.

Although no correlation was found between the average poliomyelitis death rates, case rates, and the degree of housing sanitation, it was found that there appears to be evidence of correlation between the average poliomyelitis paralytic case rates and housing sanitation, i.e., the poorer the housing sanitation of parishes, the more likely, in the Louisiana experience, are more of these parishes expected to show lower paralytic case rates (Tables 16, 17, and 18.). Since housing sanitation and income are usually factors which reflect each other, it is difficult to explain why housing sanitation shows no correlation with poliomyelitis death rates while paralytic poliomyelitis does. This could be explained by the accepted premise that poor sanitation confers immunity against poliomyelitis early in life, and therefore case rates, in this instance paralytic case rates, may be lower than under better sanitary conditions.

Summary

There is a need to continue increasing the efforts to obtain laboratory diagnosis for all poliomyelitis cases and deaths, and to improve the techniques of laboratory diagnosis and getting specimens to the laboratory in fresh state.

Although it appears that two injections of vaccine give similar reductions of paralytic cases as do three injections, it is valid at this stage of the control program to consider three injections as a completed series rather than two injections, due to the possible variability of the antigenic character of the vaccine for the three types of poliomyelitis viruses. For the same reason, it is also a valid procedure to give the fourth or booster injection a year after the completed series.

There is a need to step up our efforts for immunizing a much greater proportion of the population in the age-group below 20 years, and also the 20 to 40 year age-group.

The study of the relation of the ecological factors to poliomyelitis should be continued in order that other control measures might be revealed and pin-pointed for program planning.

Antipoliomyelitis Serum: Prospects for a Therapeutically Effective Serum*

• Owing to the sudden death of Dr. Fasting from a heart attack this manuscript has been annotated by Dr. E. C. Rosenow.†

GEORGE F. FASTING, M. D.
New Orleans

THE present unsatisfactory status of treatment of virus diseases of man and animals justifies a re-examination of the possibilities of using specific immune sera as a main or component part of treatment.

The adaptation of sundry viruses to tissue culture apart from egg-embryo growth has provided large quantities of virus antigen with relatively little foreign protein to complicate the antibody composition. The production and availability of the Salk "virus" vaccine against poliomyelitis presents the opportunity of testing the above concept. Failure to produce an effective therapeutic serum against poliomyelitis with either the Salk vaccine, or the living poliomyelitis virus from tissue culture, might reveal the difficulties to be encountered in producing antisera with other virus preparations.

The problem of preparing a specific immune serum against poliomyelitis by the use of the Salk vaccine was submitted for opinion to two sources presumed to be authorities on the question.

Opinion from Council on Pharmacy, AMA

The reply obtained from a consultant of the Council of Pharmacy of the American Medical Association was as follows:

"Prolonged treatment of large animals with the virus of poliomyelitis, to which they are unsusceptible, has not in the past yielded serums of proven value in the treatment or prophylaxis of the disease. Our present knowledge and past experience would not lead us to expect that such serums, made with the Salk vaccine, would be of use in treatment, and one would hesitate to use a foreign (nonhuman) serum for prophylaxis when gamma globulin is available."

Opinion from National Foundation

The following excerpts are taken from a reply from the research division of the National Foundation for Infantile Paralysis:

"It is possible to produce poliomyelitis antisera in animals. Such antisera were prepared and used successfully in the treatment of epidemic poliomyelitis [1] long before the development of the Salk vaccine.

"High potency monkey antiviral sera showed no therapeutic effect in experimental poliomyelitis in monkeys.

"There is no good evidence that antiviral serum therapy is of any benefit whatever if it is started after the illness of poliomyelitis has developed."

The results in prevention and treatment by Rosenow with the poliomyelitis antistreptococcal horse serum were highly favorable especially when given before or at the onset of paralysis.[2]

"Paralysis may develop or grow more severe in the presence of antibody."

However, this did not occur in poliomyelitis during treatment with the poliomyelitis antistreptococcus horse serum.[2]

The statements offered by the two con-

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sultants might have been accepted without challenge but for the fact that certain clinical and immunological data were published some twenty-five and thirty years ago.[1, 2] These very important data seemingly have been ignored. They should have been taken into account by investigators to effect a possible success in experimental and clinical serotherapy.

"Residual Antigen" in Immune Sera

In the past "antipoliomyelitis" serum was obtained either from convalescent victims, from animals repeatedly inoculated with brain-virus mixture, and from horses immunized with the streptococcus isolated in studies of poliomyelitis.[1, 2] Use of the brain-virus antigen was followed by production of brain antibodies of various intensities. Such immune sera in common with convalescent sera from monkeys and man indicated the presence of a factor which has not received due recognition. This factor may be called "residual antigen." The co-existence of "residual antigen" (original antigen and its derivatives formed after inoculation) with related specific antibodies in any immune serum may at first appear to be a strange concept. Yet, such co-existence explains in part the previously quoted statement: "Paralysis may develop or grow worse in the presence of antibody."[1] How, otherwise, is one to explain the presence of specific antibodies with disease symptoms in chronic brucellosis, tularemia and other diseases? The element of disease, has, in fact, *not* been "neutralized" by the antibodies which served the diagnostic requirements.

It is known that "normal" and immune sera carry pyrogenic elements. These appear as lipoidal substances usually linked to the alpha and beta globulins. The removal of these pyrogens and of the "residual antigens" becomes then a paramount problem in preparing immune sera for therapy.

Felton's Method

The ready removal of the noxious elements and the simultaneous separation of immune body carrying gamma globulin can be accomplished with certain immune horse sera by diluting one part of the serum with ten parts of weakly acidulated water; the pH of the final mixture should not drop below 6.5. The procedure was introduced by Felton[3, 4] some thirty years ago in the preparation of the original pneumococcal antibody solution. The product was "Council Accepted" for treatment of lobar pneumonia.

Unfortunately, Felton's method was later modified to cause total precipitation of all globulins with sodium sulfate; the globulins were then fractionated, but a small amount of pyrogenic element remained in the final product.

In accordance with present day terminology Felton's antibody solution should be classified as a specific equine gamma globulin. The clinical results with Felton's antibody solution were generally well known and established in the years preceding the use of "sulpha" drugs. Our present concern with this product is twofold: (1) that an equine source need not be a deterrent to the use of a specific gamma globulin to be employed in man: and (2) the dilution method is a means of removing a large part of antagonistic elements from the antibodies in certain immune sera.

Felton's earlier method was modified by this author to incorporate 5 per cent ether in the acidulated diluent prior to adding the serum.[3, 4] The effect of adding ether was to lower surface tension which resulted in very rapid precipitation of the gamma globulin. With this method it would be possible for most hospitals to prepare, in a few hours, adequate amounts of immune globulins from suitable antisera for therapeutic administration.

Use in Chronic Ulcerative Colitis

Clinical trials were made at the Mayo Clinic on 50 paients with well-established chronic ulcerative colitis.[5] An equine gamma globulin specific against a strain of streptococcus was prepared by the above method and was then injected subcutaneously daily in small amounts. Of

particular interest to our problem are the beneficial therapeutic results and the absence of complications which are in sharp contrast to experiences in similar earlier trials with the whole "mother" serum or its fivefold concentrate by the ammonium sulfate method.

Use in Acute Poliomyelitis

The gamma globulin of antistreptococcal serum prepared by Rosenow at the Mayo Foundation by immunizing horses with the poliomyelitis streptococcus was tried on patients with acute poliomyelitis.[1, 2] These streptococci were at the time of isolation of distinct serologic property and possessed a particular affinity for the anterior horns of the spinal cord of monkeys, rabbits, and guinea pigs on intravenous injection. Recent unpublished experiments indicate that specially preserved suspensions of these streptococci were agglutinated by the serum prepared in horses with the Salk vaccine and that other streptococci similarly preserved were not agglutinated.

The prompt suppression of fever and amelioration of symptoms in the poliomyelitis cases reported by Rosenow [2] are difficult to ascribe to a "foreign protein" reaction as this term is generally understood but were due to specific neutralization of toxic products of the virus and the neurotropic streptococcus.

Of considerable significance was the observation made by Rosenow that in persons having epidemic poliomyelitis and in monkeys that developed poliomyelitis following inoculation of the poliomyelitis virus the special equine whole antiserum and the gamma globluin fraction of such antiserum produced, on intradermal injection, an erythematous local reaction within fifteen minutes indicating circulating poliomyelitis streptococcal antigen.

If, after such a test, the patients having poliomyelitis were given a therapeutic injection of the poliomyelitis antistreptococcal serum or the corresponding solution of gamma globulin then after twenty-four hours a repeat intradermal test gave a far lesser or no reaction.[6] In this respect the cutaneous reaction paralleled the Schultz-Charlton tests in scarlet fever.

Serum from Salk Vaccine

With the foregoing information in mind the problem of immunizing horses with the Salk vaccine was undertaken. Liberal quantities of the vaccine were supplied by a noted pharmaceutical firm. The following observations have been made:

1. Repeated injections of the Salk vaccine in the horse have resulted in a high titer serum for the three main types of poliomyelitis virus in the vaccine.

2. The toxicity of the resulting serum for monkey kidney tissue did not exceed a 1:2 dilution. This is considered to be normal for any normal horse serum.

3. Subsequent inoculations with a mixture of live poliomyelitis viruses resulted in a toxicity of the immune serum approximating a 1:100 dilution.

4. Precipitation of the gamma globulin from the toxic serum by the acidulated water dilution method at pH 6.8 yielded a product shown to be *nontoxic* when tested against HeLa and monkey kidney tissue cells.

5. A trial bleeding of the above horse, one hundred and four days after the bleeding that yielded the toxic serum, gave a *nontoxic* serum.

6. Cutaneous reactions to skin tests in patients having poliomyelitis were marked to the gamma globulin fraction and minimal to the whole "mother" serum.

Having achieved a certain measure of technical success in producing and refining an antipoliomyelitis serum, we felt compelled to project the problem into the phase of therapeutic usefulness.

Therapeutic Trial

Patients having poliomyelitis were given subcutaneous injections of 25 milligrams of nitrogen in the respective globulin at one injection. Compared with serotherapies in other diseases in the past, the stated amounts would seem rather small. If one may assume from the skin tests carried on in the poliomyelitis patients that a specific gamma globulin is

capable of rapid penetration in the skin, may it not be inferred that the subcutaneous route of administering the gamma globulin should be adequate in therapy?

The gamma globulins employed in the treatment of poliomyelitis patients were prepared as needed or stored in the cold for only a couple of weeks. Freshly prepared specific gamma globulins may be of great value in therapy. The precipitation of the gamma globulin entails the loss of protective colloids present in the whole serum, and exposes the globulin to oxidation at an enhanced rate with known loss of agglutinins.

From the foregoing statements it should seem evident that the successful production of an antipoliomyelitis serum, as measured by conventional titration procedures, will require for satisfactory therapeutic results a process of "purification" and caution in its administration. In all probability a similar view must be held for other antiviral sera as they become available. It is hoped that clinical trials will soon confirm the usefulness of the specific gamma globulin from an immune equine serum resulting from the immunization with the Salk virus vaccine comparable to the use of poliomyelitis antistreptococcal serum.[1, 2]

Summary

Antipoliomyelitis serum has been produced in the horse with the Salk vaccine. The "dilution" procedure permits harvesting in a few hours the precipitate of the immune body carrying globulin. Toxic elements from the employed antigen and "inhibitory" elements in the whole serum are largely removed by the direct "dilution" technic. A "specific" gamma globulin against poliomyelitis can thus be made available for the treatment of epidemic poliomyelitis.

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Health of the City

In our editorial comments upon the health of this city, in the last number of the Journal, we remarked that serious apprehensions were entertained by many, that we should be visited by an epidemic of yellow fever. We are gratified, however, to be able to state that although fever prevailed to a considerable extent, yet it was mild, and amenable to treatment, and for this reason, as well as on account of the comparatively small number attacked, the Board of Health declared it not to be an epidemic; besides the yellow fever, another affection called the **Dengue** also made its appearance in our midst; in its onset, it resembled in many of its features the yellow fever. * * * Previous attacks of fever did not exempt persons from an attack of the **Dengue**. We believe but a few if any cases proved fatal, as the affection passed off in a few hours, or at most a few days, when opposed by mild cathartics, tepid drinks, hot foot baths, and the sulphate of quinine. It disappeared with the yellow fever.

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Editorial

The Treatment of Narcotic Addicts and the Control of Narcotic Traffic

About two years ago, the topic stated in the title was discussed in these columns.[1] The opposing viewpoints were presented. They were not entirely divergent in theory, but far apart in the practical handling of the problem. On the one hand, it was stated that drug addiction should be treated as an illness and not as a crime; that in such a situation medical psychiatric and social services should be made available to addicts. The other point of view was that 80 per cent of addicts are criminals first and become addicts in association with other addicts; that addicts cannot be cured on an ambulatory basis and that treatment in the two special hospitals for narcotic addiction, even though maintained for a sufficient time, was followed by relapse on the part of approximately two thirds. In such a situation prevention and control of addiction, together with the control of narcotic traffic, necessarily become a matter of legal and administrative procedure at the point where effectiveness of medical knowledge stops. This position still appears to be the one which organized medicine should support in the public interest.

Consideration is again being given at this time to the general problem following the final report of the Joint American Bar Association, American Medical Association Committee on Narcotic Drugs. A joint committee was formed "to explore the possibilities of a jointly conducted study of the narcotic drug traffic and related problems." Its conclusions were two: "That the present methods of dealing with narcotic addiction and narcotic addicts raised questions which are urgently in need of study, and that the legal and medical professions equally concerned can most fruitfully pursue the subject in close cooperation through their respective associations." The recommendation was a corollary to the two conclusions, namely, that the two organizations continue the study of narcotic traffic and related problems. In support of its conclusions and recommendations, the report of the joint

committee makes numerous observations. They felt that additional experience and data were needed to appraise alternative methods of controlling narcotic addiction and dealing with the drug addict.

Additional research in five major areas was suggested as follows:

"1. An experimental facility for the out-patient treatment of drug addicts, to explore the possibilities of dealing with at least some types of addicted persons in the community rather than in institutions.

"2. An extensive study of relapse and causative factors in drug addiction.

"3. The development of sound and authoritative techniques and programs for the prevention of drug addiction.

"4. A critical evaluation of present legislation on narcotic drugs and drug addiction.

"5. A study and analysis of the administration of present narcotic laws."

Consideration of these several proposed fields of investigation calls for comment. The establishment of an experimental facility for the out-patient treatment of addicts has been tried in the past in this country and in various others, notably here in the period between 1919 and 1923. The result was so bad in most areas that the plan was condemned by the A.M.A., and ultimately abandoned. In other countries it has been equally a failure.

Research into the causes of relapse and inquiry into the possible programs for the prevention of drug addiction have been undertaken under suitable auspices for some time past. Ultimately, the findings in such investigations are going to be helpful.

The fourth and fifth probably have a bearing on the committee's attitude toward that phase of the federal narcotic laws administration that prevents the prescribing and dispensing of narcotics to drug addicts merely for the purpose of gratifying addiction. This matter has been considered and discussed since the first enactment of the Harrison Narcotic Act. It has also recently had thorough investigation independently by two committees in Congress. The conclusions arrived at on the part of the majority of those who are familiar with the problem, and by the two Congressional committees, were that the present laws and the current interpretation of the administration serve the best public interest.

Further observations by the joint committee, while admitting "zealous law enforcement efforts have unquestionably played a part in reducing drug addiction —and will indisputably continue to be required—in curbing the ilicit drug traffic" raised the question as to whether the law enforcement policies urged by the U. S. Narcotic Bureau provide the full answer to the problem, and whether severe jail and prison sentences are the most rational way of dealing with narcotic addicts.

The Committee pleads that the addict, because of his physical and psychological dependence upon drugs and because of his frequently abnormal personality patterns, would be as much of a subject of concern to medicine and public health as to those having to do with law enforcement. The comment, however, from those whose experience in this field is extensive, is that where medical and psychological approaches to the problem fail there is no other way to deal with the addict or peddlar, or both, than through the proper administration of laws which have been found to be effective.

The joint committee report was presented to the House of Delegates of the American Medical Association by the Board of Trustees in the June 1959 meeting. The report of the reference committee, which was approved by the House, was as follows:

"Your reference committee wishes to congratulate the Joint AMA-ABA Committee on Narcotic Drugs on its long, tedious, and fruitful work on the broad subject of narcotics. The Committee is to be commended for its efforts and results. Since the recommendation is for continued study under separate, already established councils of the AMA and ABA, your reference committee urges its imple-

mentation as soon as possible."

This action of the committee is interpreted as not adopting or approving the joint committee's report, except insofar as it pertains to further study. This means that the House did not depart from its previously taken position of objecting to a plan of treating drug addicts by continuing to supply regular dosage of the drug of addiction nor did it revoke its long standing policy of opposition to the so-called ambulatory treatment.

The position, therefore, of organized medicine seems to be that of disapproving clinics for ambulatory treatment, and favoring adequate administration of effective law, and further investigation into the fundamentals of the drug addiction problem. Caution and conservatism in this field are to be commended.

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[1] Editorial: Proposed changes in the medical and legal handling of narcotic addiction, J. Louisiana State M. Soc. 109:267, (July) 1957.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

INCREASE IN BENEFITS COVERED BY HEALTH INSURANCE POLICIES

Benefit provisions in health insurance policies covering hospital and surgical care have been improved steadily throughout the 1950's, the Health Insurance Institute reported.

In 1951, a survey of some 101 insurance companies showed that the top daily hospital allowance offered by 89% of these companies averaged $8 or less, the Institute said. At that time, only 5% of the total number of companies surveyed offered a policy paying $10 a day or more.

Three years later, with the added experience which insurance companies gained with this form of health insurance protection, the situation had changed. In 1954, a survey of 186 insurance companies disclosed that 72% offered policies with hospital benefits of $15 a day or more. Some 11% offered policies at $20 a day and 4% offered $25 daily or more. Only three of the companies surveyed that year had a maximum daily hospital benefit of $8.

The trend toward more adequate daily hospital benefits had continued, the Institute found.

A recent review of 188 insurance companies indicated that 93% offer maximum daily hospital benefit of $15 or more. This same analysis, reported the Institute, showed that 32% of the surveyed companies offer $20 a day or more, and 17% of the companies offer upwards of $25 a day. In addition, at least three companies have policies with hospital benefits of $30 a day or more.

The maximum duration of stay in the hospital also has been extended the Institute stated.

The 1951 survey of 101 companies indicated that 59% of the companies offered a maximum of 90 days of hospitalization a year and that 12% of the companies offered more than 120 days. The 1954 study of 181 companies disclosed 51% offered 90 days and 20% offered 120 days or more. A more recent analysis of 188 companies indicated that about 32% would write 120 days or more, and that 18% would cover hospitalization up to a year.

The broadening of benefits in available health insurance policies also holds true for surgical expense coverage, said the Institute. Among 183 companies surveyed in 1954, some 16% offer maximum surgical benefits of $300 or more. Currently, of 188 companies analyzed, 72% offer a surgical maximum of $300 or more.

GOOD WORK OF THE SOCIETY

Last month it was called to the attention of the Society that the American Legion at its state meeting of 1958 had endorsed a resolution to enable chiropractors to practice in VA hospitals. Doctor Ralph Riggs did excellent work after this information was brought to his attention and contacted the local American Legion, which he found most cooperative. He and Doc-

tor Broox Garrett met with some members of the American Legion here and valuable information was obtained. This information was transmitted to Doctor Robyn Hardy, President of the Louisiana State Medical Society, and Doctor C. Grenes Cole, Secretary-Treasurer. By hard and excellent work on the part of the State Society, the Resolutions Committee of the American Legion met and did not endorse chiropractic care for patients in VA hospitals for 1959.

It was shown clearly that the American Legion would like very much to work in close cooperation with the doctors of the state and local societies. More doctors who are eligible for membership should take an active part in the American Legion. Doctor Riggs is to be particularly congratulated for his excellent work. Also, Doctor Broox Garrett.

The American Legion is to be commended for its attitude toward us. We should cultivate each other.

C. E. Boyd, M. D.
Fourth District Councilor
Louisiana State Medical Society

We wish to further add to this article by Dr. Boyd that Dr. Cole, our Secretary, although on his vacation at his country home, spent an entire day including part of the night in contacting members of the Resolutions Committee of the State Branch of the American Legion requesting that they refuse to approve any resolution recommending to the national group that Chiropractors be allowed to practice in Veterans' Administration Hospitals. We are happy to report that all eight members of this Committee, in addition to the State Commander, assured us that they would definitely oppose such a resolution, and at their meeting in Baton Rouge they refused to support such a resolution suggested by the chiropractors.

It is our concerted belief that, if more of our doctors who are eligible for membership, or who are already members of the Legion would attend the meetings of the Legionnaires that a situation like this would never develop or if it did come up that such a move would be nipped in its bud, and this last minute rush of contacts would be avoided. We strongly urge that more of our doctors attend their Legion meetings, thereby protecting their own and the profession's interest.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Ascension	Third Tuesday of every month	
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Lafayette	Second Tuesday of every month	Lafayette
Morehouse	Third Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LOUISIANA ACADEMY OF GENERAL PRACTICE

Thirteenth Annual Scientific Assembly

The Louisiana Academy of General Practice will hold its Thirteenth Annual Scientific Assembly on October 20-22, 1959, at the Capitol House in Baton Rouge, Louisiana, it was announced by Dr. Francis I. Nicolle of New Orleans, Secretary of the organization.

The Congress of Delegates of the Academy will be in session on the morning of Tuesday, October 20. Speaker of the Congress is Dr. Eldredge L. Carroll of Columbia and vice-speaker is Dr. John G. McClure of Welsh. The scientific sessions will begin on the afternoon of Tuesday, October 20 in the Ballroom of the Capitol House.

The scientific portion of the program has been accepted for eleven hours Category I credit by the American Academy of General Practice which can be applied toward the 150 total hours of postgraduate study needed for each three-year period of continuing membership in the Academy.

Dr. Rafael C. Sanchez of New Orleans, Chairman of the Committee on Scientific Assembly, has arranged for a series of interesting and informative speakers, and in addition special medical films to round out the scientific program.

Dr. Frank G. Rieger of Baton Rouge is Convention Chairman and his committee chairmen include Dr. J. E. Williams, Dr. P. M. Castro, Dr. H. B. Holden, Dr. Melvin D. Robinson, Dr. G. C. Black, Dr. T. Y. Gladney, Dr. C. R. Chestnutt, Dr. R. C. A. Bock, Dr. M. O. Holt, Dr. J. W. Lorio, Dr. F. A. deJean and Dr. J. M. Hopper, all of Baton Rouge; Dr. C. B. Smith of Baker and Dr. R. J. Berthelot of Zachary.

Convention Chairman for the Woman's Auxiliary is Mrs. Melvin D. Robinson of Baton Rouge. Co-Chairman is Mrs. Mortimer F. Currier of Plaquemine.

On Wednesday, October 21, the Louisiana Academy will hold its annual installation dinner, at which time Academy officers for the coming year will be presented. The installing officer will be Dr. John G. Walsh of Sacremento, California, President-elect of the American Academy of General Practice. Dr. Thomas H. Nelson and Dr. Henry D. Pope, Jr., 1959 Mead Johnson Award winners for Graduate Training in General Practice, will be guests of the Academy at this function. Both of these doctors are at present residents at the Huey P. Long Charity Hospital in Pineville. Invocation at installation dinner will be given by the Rt. Rev. Monsignor Herman P. Lohman, Pastor of St. Joseph's Catholic Church in Baton Rouge.

POSTGRADUATE COURSES ON DISEASES OF THE CHEST

The Council on Postgraduate Medical Education of the American College of Chest Physicians will present the following Postgraduate Courses this fall:

14th Annual Course, CLINICAL CARDIOPULMONARY PHYSIOLOGY
Edgewater Beach Hotel,
Chicago, October 5-9

12th Annual Course on DISEASES OF THE CHEST
Park Sheraton Hotel,
New York City, November 9-13

5th Annual Course on DISEASES OF THE CHEST
Ambassador Hotel,
Los Angeles, December 7-11

The course on Clinical Cardiopulmonary Physiology is the second to be presented by the Council on this timely subject. The New York City and Los Angeles courses will offer the most recent advances in the diagnosis and treatment of chest diseases—medical and surgical.

Tuition for each course is $100 including round table luncheon discussions.

Further information may be obtained by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

FORTHCOMING MEETINGS

The Tennessee Valley Medical Assembly will meet at the Read House in Chattanooga, Tennessee, on September 28 and 29, 1959.

The fourth national cancer conference will meet in Minneapolis, September 13-15, 1960. For further information, contact Medical Arfairs Department, American Cancer Society, 521 W. 57th St., New York 19.

TULANE LIBRARIAN RETIRES

Miss Mary Louise Marshall, librarian of the Rudolph Matas Medical Library and professor of medical bibliography at Tulane medical school, retired in September following her return from a three month tour as library consultant to the medical schools in Colombia, South America. Miss Marshall is the ninth Tulane medical faculty member to visit Colombia as part of the three year old Tulane-Colombia project. Under its terms Tulane cooperates with the seven medical schools of Colombia as advisor on medical education and research. Miss Marshall has served as Tulane medical librarian for 32 years and was appointed professor of medical bibliography in 1949.

Succeeding Miss Marshall is William D. Postell, who has been librarian and professor of medical bibliography at the School of Medicine of Louisiana State University since 1938. Mr. Postell is a past president of the Medical Library Association, has received the Marcia C. Noyes Award of the Association for outstanding contributions to medical librarianship, and is the author or editor of four books and 58 professional articles, among other distinctions.

WEIGHT REDUCING AGENT TERMED INEFFECTIVE

A widely used appetite suppressent has been found to be ineffective in helping obese persons, a group of eastern scientists reported recently.

The suppressent—phenylpropanolamine—is a common ingredient of weight-reducing drugs which are offered to the public without prescriptions. It has been estimated that about 100 million dollars is spent annually for such drugs, the scientists said.

Their report appears in the June 27 Journal of the American Medical Association.

Headed by Dr. Joseph F. Fazekas, Washington, D. C., the group undertook the study to determine the merits of the non-prescription drug as compared with dextro amphetamine, a

prescription drug with known appetite suppressing qualities.

The authors said that any effective weight-reducing program must be administered by a physician who understands the psychological factors which are encountered by persons undergoing treatment.

AMERICAN FRACTURE ASSOCIATION

The 20th Annual Meeting of the American Fracture Association and a one day postgraduate course on fractures will be held at the Roosevelt Hotel in New Orleans, November 1 through 4. Both events are acceptable for credit with the American Academy of General Practice and the Louisiana Academy of General Practice. The Postgraduate Course, sponsored by the Division of Graduate Medicine of Tulane School of Medicine, will grant four and a half hours of credit in category one for registrants. Tuition fee is ten dollars. Participants in the program of the annual meeting include Dr. Howard J. Goldstein, Montgomery, Alabama; Dr. W. Courtney Seagle, Urbana, Illinois; Dr. Jack Wickstrom, New Orleans; Dr. A. H. Biehr, St. Louis; Dr. John B. Erich, Rochester, Minnesota, and Dr. Sidney N. Eichenholtz, Yonkers, New York. An optional tour of the National Leprosarium, U. S. Public Health Service Hospital at Carville, La., will be conducted on November 4. General Chairman for the meetings is Dr. Elias N. Kaiser, Montgomery, Alabama.

SYMPOSIUM
CHANGING CONCEPTS IN MEDICINE

Deborah Hospital announces the Second International Symposium on Changing Concepts in Medicine (Congenital Heart Disease) to be held at the Bellevue-Stratford Hotel in Philadelphia, on April 28, 29, 30, 1960.

The Symposium is being organized under the direction of a National Committee headed by Doctor Charles P. Bailey, Chairman of the Medical Board of Deborah Hospital. Eminent clinician-educators will lead panel discussions encompassing all aspects of congenital heart conditions and the selection of patients for surgical corrections.

Inquiries about the Symposium should be addressed to: Dr. Charles P. Bailey, Deborah National Office, 901 Walnut Street, Philadelphia 7, Pennsylvania.

CARIBBEAN CRUISE

The University of Tennessee College of Medicine will give physicians an opportunity for postgraduate education while sailing the Caribbean for eight days next January.

Wallace Mayton, director of the Postgraduate Department at UT, said the eight-day cruise will leave from New Orleans January 15 and visit Nassau in the Bahamas; Havana, Cuba; and Montego Bay, Jamaica, spending four days in these ports of call.

He said the cruise is made possible through the cooperation of the Tennessee Academy of General Practice, the State's Association of General Practitioners of Medicine.

The physicians will sail aboard the TS Ariadne, a vessel owned by the Hamburg American Line in Hamburg, West Germany. The Allen Transfer Service in New York City made arrangements for the voyage. The physicians will spend about five days at sea and each morning from 8:30 until 12:30 seminar sessions will be held. The sessions will include lectures on a variety of medical topics, lantern slide projection, and motion pictures on surgical procedures of interest to general practitioners. The physicians may be accompanied by their wives.

Twenty hours of postgraduate credit is offered by the American Academy of General Practice to those who make the voyage. The cost of the trip will be approximately $350.

The faculty will include:

Dr. M. K. Callison, dean of the College of Medicine; Dr. Harwell Wilson, chief of the Division of Surgery; Dr. R. H. Kampeier, professor of medicine, Vanderbilt University; Dr. I. Frank Tullis, chief of the Division of Medicine; Dr. L. W. Diggs, head of the Department of Medical Laboratories; Dr. James G. Hughes, professor of Pediatrics.

Further information may be obtained from the Postgraduate Department of the University at 62 South Dunlap.

STUDY SHOWS EXTENT OF VENOM DEATHS

Nearly as many Americans die from bee stings as from rattlesnake bites, a new study has shown.

In addition, they die from the bites and stings of wasps, hornets, yellow jackets, ants, cottonmouth moccasins, coral snakes, scorpions, spiders, and sting rays.

Dr. Henry M. Parrish, University of Vermont College of Medicine, Burlington, studied the death certificates of all persons in the United States who died from bites and stings of venomous animals and insects during the five-year period 1950 through 1954. The study is reported in the August issue of Archives of Internal Medicine, published by the American Medical Association.

There were 215 deaths, with an average of 43 a year, and an average death rate of 0.28 per 1 million population per year.

The Hymenoptera—bees, wasps, hornets, yellow jackets, and ants—killed 86 persons (40 per cent), while poisonous snakes—rattlers, cottonmouth moccasins, coral and unidentified—

killed 39 persons (18 per cent). Rattlesnakes killed 55 persons, while bees killed 52.

More children died of snake bite than from bee stings, apparently because bee sting deaths are actually severe allergic shock reactions and the person must previously have been sensitized to the insect venom. In addition, children get a proportionately larger amount of snake venom according to body weight than do adults, Dr. Parrish said.

Other animals and insects causing death were scorpions (5), spiders (39), Portugese man-of-war (1), and sting ray (1). Twelve other deaths were caused by unknown animals and insects.

Centipedes, millipedes, Gila monsters, poisonous fishes, copperhead moccasins, and tarantulas were not reported to produce any deaths. This does not mean that they are not poisonous, but it does indicate that they are not a major source of human deaths in the United States, Dr. Parrish noted.

He believes that previous estimates of the incidence of bites and stings by venomous animal are too low. Very likely, he said, deaths in the United States from venomous animals are attributed to other causes on death certificates. This is especially true of Hymenoptera-caused deaths, since these allergic shock reactions are easily misdiagnosed, he said.

FEVER NEEDS FOOD, WATER, ACCORDING TO DOCTORS

A fever must be watered and fed, not starved, two Chicago physicians reported.

Fever leads to dehydration and the breakdown of body tissues. At high temperatures, the body cells work faster and break down more rapidly. To replace them, the body needs food and water.

Drs. Rachmiel Levine and Sidney Cohen of the Medical Research Institute of Michael Reese Hospital gave their advice in an interview reported in the forthcoming (September) Today's Health, published by the American Medical Association.

When a fever reaches 103 or more, it becomes dangerous, they said. It leads to the malfunctioning of the central nervous system and heart because every degree of fever makes the heart beat faster, and over a certain beat rate, fever becomes injurious to the heart.

The seriousness of a fever depends on age, persistence, and variability, the doctors said. A child's body temperature will react more drastically with a fever than an older person's. The older a person grows the less drastic and slower are his reactions to fever-causing agents.

Whether a fever persists day after day whether it has peaks at certain times a day influence its seriousness.

Fever, according to the doctors, is a symptom or indicator; it is not a disease. It may be associated with infections, cirrhosis of the liver, gout, neoplastic diseases, vascular accidents, pregnancy, food intake and excessive fatigue. It may be caused by hypersensitivity to drugs.

There is no general remedy for fever, although aspirin is often given, they said. Fever induced by infection is reduced by eliminating the infectious germs.

Fever should be considered as an indicator that something may be wrong and should be thoroughly investigated, they said. The best way is to check with a doctor.

However, a person should not become obsessed about slight elevations in body temperatures, once examination has shown no cause for the slight rise in temperature. Some persons just normally have a higher than "normal" temperature all the time.

CHOLESTEROL ROLE IN HEART DISEASE STILL UNKNOWN

The significance of lowered blood cholesterol levels in the prevention and treatment of heart disease is not definitely known, according to the American Medical Association's Council on Foods and Nutrition.

Methods of manipulating the blood levels of cholesterol have become of utmost interest, and because their importance is unknown confusion has resulted, the council said in a report in the Aug. 29 A.M.A. Journal.

In an attempt to keep physicians informed of current knowledge about cholesterol, the council presented the views of five foremost researchers in the field.

"Of all the chemical compounds that are measured in clinical laboratories, there is none about which more has been written and about which less is understood than cholesterol," according to Dr. Lawrence W. Kinsell, Institute for Metabolic Research, Highland-Alameda County Hospital, Oakland, Calif.

It is well established, he said, that, statistically, elevated levels of cholesterol are found in association with atherosclerosis (one form of hardening of the arteries). It seems reasonable, therefore, to believe that measures directed toward lowering the levels may work in a desirable way.

However, since cholesterol is a normal essential part of the human body, "it is obvious that attempts to 'get rid of' this compound would be both unphysiological and impossible," Dr. Kinsell said.

The objective, then, must be to achieve normal cholesterol metabolism with consequently normal blood levels in the hope that such a program will prevent abnormal deposits of cholesterol in the blood vessels, he said.

Among the methods mentioned by the physicians are the use of diet, such drugs as nicotinic acid, estrogens, and increased exercise.

BOOK REVIEWS

Medical Radiation Biology; by Friedrich Ellinger, M.D. Springfield, Illinois, Charles C Thomas, 1957, pp. 945. Price $20.00.

The author, who has done work in this field for many years and who is presently chief radiation biologist and head of the pharmacology division at the Naval Medical Research Institute, has written a book of timely interest and of considerable contemporary importance. It is his intent to indicate "the rationale on which the role of radiation as a health hazard and as a therapeutic agent is based".

The initial portion of the book is of an introductory nature. Radiations are discussed in a general way, along with their mode of action, as well as some of their pertinent chemical and cellular effects. Also included in this section is a discussion of radiosensitivity. The second and major portion of the book is devoted to the effects of radiant energy upon the various organs and organ systems of the body and various disease processes, including malignant neoplasms. Material pertaining to radiation as a health hazard is also presented. The last sections of the book are devoted to the biologic effects of ultra-violet, infra-red and visible light. There is a very complete, well organized table of contents, but no index. Forty-six hundred references are listed.

ROBYN HARDY, M.D.

Chemosurgery in Cancer, Gangrene and Infections; by Frederick E. Mohs, M. D. Charles C Thomas, Springfield, Ill., 1956, pp 305, Price $13.50

The author has devoted many years in the clinical evaluation of the chemosurgical treatment of cancer. He has accumulated a considerable amount of information on this subject with photographs and has expanded it to include gangrene and infections. The book presents a practical work description of the technics employed and shows this conservative procedure to be fairly reliable. He has thoroughly documented statistics on the five year end results in a goodly series of cases. This book is a must for anyone interested in treating advanced visible cancer.

CHARLES C. ABBOTT, M. D.

Ear, Nose and Throat Dysfunctions Due to Deficiencies and Imbalances; by Sam E. Roberts, Springfield, Illinois, Charles C Thomas, 1957, pp. 323, price $8.50.

This text deals with the author's own observations and philosophies concerning various diseases rather than with established facts. Much of its content is highly speculative if not fanciful. The author places much emphasis on therapy with a balanced diet and sex hormones for various disorders such as vascular headaches, Meniere's disease, acoustic nerve dysfunction.

The most useful function of this book to the otolaryngologist is to remind him that the patient's complaints may originate outside the ear, nose and throat system.

The author's expressive style is quite pleasant.

L. J. RUTLEDGE, M. D.

Atlas of Clinical Endocrinology; by Lisser-Escamill. St. Louis, C. V. Mosby, 1957, pp 476, Price $18.75

This excellent atlas will appeal to all physicians, for endocrinologic problems appear in all aspects of medical practice. In addition to numerous first-rate pictures, the book includes an abbreviated text with all necessary information for diagnosis and therapy. There is a brief but well selected bibliography.

V. J. DERBES, M. D.

A Textbook of Histology; by Alexander A. Maximow and William Bloom. (7th edition) Philadelphia, Pa. W. B. Saunders Co., 1957. pp. 628, 631 illus., Price $11.00.

Alexander Maximow, who died in 1928, had not completed his manuscript of the original edition of this long-favored textbook. William Bloom brought the manuscript to completion and since has carried the book through revision after revision. Seven editions and widespread use of this text through a quarter-century attest to its quality. The text is so generally familiar that a full review is unnecessary. The present edition maintains the standard that had been set from the first: the text matter is authoritative and clearly written; the illustrations are well chosen and excellent technically. Again Dr. Bloom has called upon specialists to contribute to the revision. It is noteworthy that additional emphasis has been placed upon electron microscopy, histochemistry and other phases of histology that are rapidly expanding.

HAROLD CUMMINS, PH.D.

PUBLICATIONS RECEIVED

The C. V. Mosby Co., St. Louis; Synopsis of Ophthalmology, by William H. Havener, M. D.; The Care of Minor Hand Injuries, by Adrian E. Flatt, M. D.; Anesthesia for Infants and Children, by Robert M. Smith, M. D.; Synopsis of Ear, Nose, and Throat Diseases, by Robert E. Ryan, M. D., William C. Thornell, M. D., and Hans von Leden, M. D.

Prentice-Hall, Inc., Englewood Cliffs, N. J.: What Next, Doctor Peck?, by Joseph H. Peck, M. D.

Charles C Thomas, Publisher, Springfield, Ill.: Nutritional Diagnosis, by Grace A. Goldsmith, M. D.